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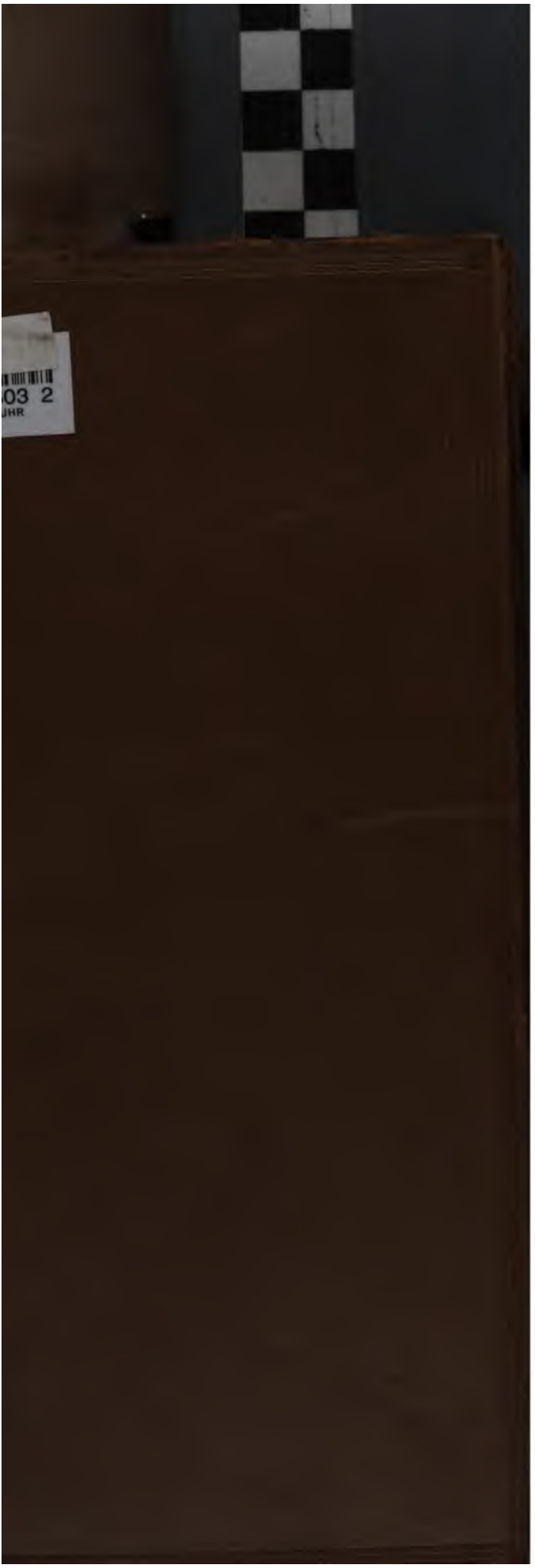
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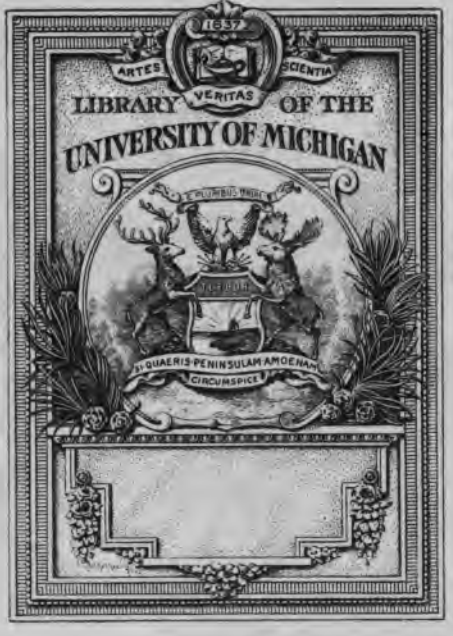
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T.—Treasurer.	S.—Secretary.
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AUGUST 1853.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1841 *James Abercrombie, M.D., Cape of Good Hope.

1846 *John Abercrombie, M.D., Physician to the General Dispensary, Cheltenham; Cheltenham.

1851 *Henry Wentworth Acland, M.D. F.R.S., Physician to the Radcliffe Infirmary, Oxford.

1842 William Acton, Queen Anne-street, Cavendish-square.

1818 Walter Adam, M.D., Physician to the Royal Public Dispensary, Edinburgh.

1851 John Adams, Surgeon to the London Hospital; St. Helen's-place, Bishopsgate-street.

1852 William Adams, 5, Henrietta-street, Cavendish-square.

1818 Thomas Addison, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; New-street, Spring-gardens. C. 1826. V.P. 1837. P. 1849. C. 1853.

1814 Joseph Ager, M.D., Great Portland-street, Portland-place. C. 1836.

1837 *Ralph Fawsett Ainsworth, M.D., Manchester.

1819 George Frederick Albert.

1839 Rutherford Alcock, K.C.T. K.T.S., China.

Elected

1826 JAMES ALDERSON, M.D. F.R.S., *Vice-President;* Physician to St. Mary's Hospital; Berkeley-square. S. 1829. C. 1848. T. 1849.

1843 CHARLES JAMES BERRIDGE ALDIS, M.D., Physician to the London and Surrey Dispensaries, and Lecturer on Medicine at the Hunterian School of Medicine; Chester-terrace, Chester-square.

1850 CHARLES REVANS ALEXANDER, Assistant-Surgeon to the Royal Infirmary for Diseases of the Eye; Cork-street, Bond-street.

1813 HENRY ALEXANDER, F.R.S., Surgeon-Oculist in Ordinary to the Queen, and Surgeon to the Royal Infirmary for Diseases of the Eye; Cork-street, Bond-street. C. 1840. V.P. 1850.

1836 HENRY ANCELL, Norfolk-crescent, Oxford-square. C. 1847.

1817 ALEXANDER ANDERSON.

1820 THOMAS ANDREWS, M.D., Norfolk, Virginia.

1813 WILLIAM ANKERS, Knutsford.

1819 PROFESSOR ANTOMMARCHI, Florence.

1825 THOMAS GRAHAM ARNOLD, M.D., Stamford.

1819 JAMES MONCRIEFF ARNOTT, F.R.S., New Burlington-street. L. 1826. V.P. 1832. T. 1835. C. 1846. P. 1847.

1817 JOHN ASHBURNER, M.D. M.R.I.A., 40, York-place, Portman-square. C. 1821.

1851 THOMAS JOHN ASHTON, Surgeon to the Blenheim Dispensary; 31, Cavendish-square.

1841 JOHN AVERY, Surgeon to the Charing-cross Hospital; Queen-street, May-fair.

1825 BENJAMIN GUY BABINGTON, M.D. F.R.S., Physician to Guy's Hospital, and Physician to the Deaf and Dumb Institution; George-street, Hanover-square. C. 1829. V.P. 1845. T. 1848.

1846 CORNELIUS METCALFE STUART BABINGTON, M.D., Physician to Queen Charlotte's Lying-in Hospital; 29, Hertford-street, May-fair.

1820 *JOHN H. BADLEY, Dudley.

1838 FRANCIS BADGLEY, M.D., Toronto, Upper Canada.

1840 WILLIAM BAINBRIDGE, Kingston, Surrey.

Elected

1836 ANDREW WOOD BAIRD, M.D., Ipswich.

1851 *ALFRED BAKER, Surgeon to the General Hospital, Birmingham.

1839 THOMAS GRAHAM BALFOUR, M.D., Royal Military Asylum; Chelsea. C. 1852.

1848 EDWARD BALLARD, M.D., Myddleton-square.

1849 THOMAS BALLARD, Southwick-place, Hyde-park.

1837 WILLIAM BALY, M.D. F.R.S., Physician to the Milbank Prison, and Lecturer on Forensic Medicine at St. Bartholomew's Hospital; Queen Anne-street, Cavendish-square. C. 1845. L. 1847. S. 1848.

1847 ANDREW WHYTE BARCLAY, M.D., Physician to the Chelsea Dispensary; Bruton-street, Berkeley-square.

1848 EDGAR BARKER, Edgeware-road, Hyde-park.

1833 THOMAS ALFRED BARKER, M.D., *Vice-President;* Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Grosvenor-street, Grosvenor-square. C. 1844.

1843 THOMAS HERBERT BARKER, Priory-terrace, Bedford.

1847 GEORGE HILARO BARLOW, M.D., Physician to Guy's Hospital; Union-street, Southwark.

1840 BENJAMIN BARROW, Ryde, Isle of Wight.

1844 WILLIAM RICHARD BASHAM, M.D., *Secretary;* Physician to, and Lecturer on Medicine and Materia Medica at, the Westminster Hospital; Chester-street, Grosvenor-place.

1841 GEORGE BEAMAN, King-street, Covent-garden.

1836 WILLIAM BEAUMONT, Professor of Surgery in the University of King's College; Toronto, Upper Canada.

1840 CHARLES BEEVOR, Berners-street, Oxford-street.

1819 THOMAS BELL, F.R.S. L.S. and G.S., Professor of Zoology in King's College, London, and Lecturer on Diseases of the Teeth at Guy's Hospital; New Broad-street, City. C. 1832.

1847 JAMES HENRY BENNET, M.D., Grosvenor-street.

1845 EDWIN UNWIN BERRY, James-street, Covent-garden.

1820 STEPHEN BERTIN, Paris.

1827 WILLIAM BIRCH, Barton, Lichfield.

1845 GOLDING BIRD, M.D. F.R.S., Assistant-Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Russell-sq.

Elected

1850 JAMES BIRD, M.D., Hyde-park-square, Hyde-park.

1849 EDMUND LLOYD BIRKETT, M.D., 4, Montague-street, Russell-square.

1851 GEORGE BIRKETT, M.D., 9, Duncan-terrace, Islington.

1851 JOHN BIRKETT, Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 6, Wellington-street, Southwark.

1846 HUGH BIRT, Morro Velhio, Minas Geraes, Rio Janeiro, Brazil; Surgeon to the Morro Velhio Hospital.

1843 PATRICK BLACK, M.D., Assistant-Physician to St. Bartholomew's Hospital, and Warden to the Collegiate Establishment at St. Bartholomew's Hospital.

1844 THOMAS BLACKALL, M.D., Physician to the Seamen's Hospital Ship 'Dreadnought;' Queen-street, May-fair.

1847 GEORGE C. BLACKMAN, M.D., New York, U.S.

1839 RICHARD BLAGDEN, Surgeon-Accoucheur, and Surgeon Extraordinary to the Queen, Surgeon in Ordinary to Her Royal Highness the Duchess of Kent; Albemarle-street, Piccadilly. C. 1847.

1840 PEYTON BLAKISTON, M.D. F.R.S., St. Leonard's-on-Sea.

1845 HENRY BLENKINSOP, Warwick.

1823 LOUIS HENRY BOJANUS, M.D., Wilna.

1816 HUGH BONE, M.D., Inspector-General of Hospitals; Edinburgh.

1810 JOHN KAYE BOOTH, M.D.

1846 PETER BOSSEY, Thomas-street, Woolwich.

1846 JOHN ASHTON BOSTOCK, 34, Clarges-street, Piccadilly.

1849 EDWARD BARONS BOWMAN, M.D., Oxford-terrace, Middleton-road, Dalston.

1841 WILLIAM BOWMAN, F.R.S., Professor of Physiology and General Anatomy at King's College, London; Assistant-Surgeon to King's College Hospital, and to the Royal Ophthalmic Hospital, Moorfields; Clifford-street, Bond-street. C. 1852-3.

1814 RICHARD BRIGHT, M.D. F.R.S., Physician Extraordinary to the Queen, and Consulting Physician to Guy's Hospital; Savile-row, Regent-street. C. 1821. V.P. 1827. P. 1837.

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1851 BERNARD EDWARD BRODHURST, Assistant-Surgeon to the Royal Orthopædic Hospital; Brook-street, Grosvenor-square.

1813 SIR BENJAMIN COLLINS BRODIE, BART., D.C.L. F.R.S., Serjeant-Surgeon to the Queen, Surgeon in Ordinary to His Royal Highness Prince Albert, Foreign Correspondent of the Institute of France, and Foreign Associate of the Royal Academy of Medicine of Paris; Savile-row, Regent-street. C. 1814. V.P. 1816. P. 1839.

1844 CHARLES BROOKE, B.A. (Cantab.) F.R.S., Surgeon to the Westminster Hospital; Keppel-street, Russell-square.

1848 WILLIAM PHILPOT BROOKES, M.D., Surgeon to the Cheltenham General Hospital and Dispensary, and Visiting Medical Officer to the Cheltenham District of Lunatic Asylums; Albion House, Cheltenham.

1842 CHARLES BLAKELY BROWN, M.B., Physician to Queen Charlotte's Lying-in Hospital, and St. George's and St. James's Dispensary; Hill-street, Berkeley-square.

1847 GEORGE BROWN, Grenadier Guards' Hospital, Rochester-row, Westminster.

1847 *ROBERT BROWN, Winckley-square, Preston, Lancashire.

1851 ALEXANDER BROWNE, M.D., Army and Navy Club, St. James's-square.

1827 M. PIERRE BRULATOUR, Surgeon to the Hospital; Bordeaux.

1823 B. BARTLET BUCHANAN, M.D.

1843 JOHN CHARLES BUCKNILL, M.B., Axminster, Devonshire.

1839 GEORGE BUDD, M.D. F.R.S., Fellow of Caius College, Cambridge; Professor of Medicine in King's College, London; Physician to King's College Hospital; Dover-street, Piccadilly. C. 1846.

1839 THOMAS HENRY BURGESS, M.D., Half-moon-street, Piccadilly.

1833 GEORGE BURROWS, M.D. F.R.S., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Cavendish-square. C. 1839. T. 1845. V.P. 1849.

1820 SAMUEL BURROWS.

1837 GEORGE BUSK, F.R.S., Surgeon to the Seamen's Hospital-ship 'Dreadnought;' Croom's-hill, Greenwich. C. 1847.

Elected

1850 JOHN STEVENSON BUSHNAN, M.D.

1818 JOHN BUTTER, M.D. F.R.S. F.L.S., Physician to the Plymouth Eye Infirmary; Plymouth.

1851 *WILLIAM CADGE, All Saints, Norwich.

1851 THOMAS CALLAWAY, Demonstrator of Anatomy at Guy's Hospital; Wellington-street, Southwark.

1852 *GEORGE CANNEY, Bishop's Auckland, Durham.

1842 HENRY CANTIS, 14, Hanover-street, Hanover-square.

1847 JOHN BURFORD CARLILL, M.D., Berners-street, Oxford-street.

1839 SIR ROBERT CARSWELL, M.D., Physician to his Majesty the King of the Belgians; Brussels.

1825 HARRY CARTER, M.D., Physician to the Kent and Canterbury Hospital; Canterbury.

1853 ROBERT BRUDENELL CARTER, Leytonstone, near London.

1818 *RICHARD CARTWRIGHT.

1820 SAMUEL CARTWRIGHT, F.R.S., Savile-row, Regent-street, and Nizell's House, near Tonbridge.

1845 SAMUEL CARTWRIGHT, Jun., Savile-row, Regent-street.

1839 WILLIAM CATHROW, Weymouth-street, Portland-place.

1845 WILLIAM OLIVER CHALK, Nottingham-terrace, New-road.

1818 RICHARD CHAMBERLAINE, Kingston, Jamaica.

1844 THOMAS KING CHAMBERS, M.D., Physician to St. Mary's Hospital; Hill-street, Berkeley-square.

1816 *WILLIAM FREDERICK CHAMBERS, M.D. K.C.H. F.R.S.; Hardle House, near Lymington, Hants. C. 1818. V.P. 1821. P. 1845.

1849 FREDERICK CHAPMAN, Richmond-green, Richmond, Surrey.

1837 HENRY THOMAS CHAPMAN, Lower Seymour-street, Portman-square.

1838 GEORGE CHAPLIN CHILD, M.D., Consulting Physician to the Westminster General Dispensary; Queen Anne-street, Cavendish-square. C. 1853.

1852 GEORGE BORLASE CHILDS, Finsbury-place South, Finsbury-square.

1849 WILLIAM FRANCIS CHORLEY, M.D., Physician to the St. Marylebone Dispensary; 3, South Molton-street, Oxford-street.

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1842 WILLIAM DINGLE CHOWNE, M.D., Physician to the Charing-cross Hospital; Connaught-place West, Hyde-park. C. 1853.

1847 BENJAMIN CLARK.

1839 FREDERICK LE GROS CLARK, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, St. Thomas's Hospital; Consulting Surgeon to the Western General Dispensary; Spring-gardens. S. 1847.

1827 SIR JAMES CLARK, Bart., M.D. F.R.S., Physician to the Queen, Physician in Ordinary to his Royal Highness Prince Albert, and Consulting Physician to his Majesty the King of the Belgians; Brook-street, Grosvenor-square. C. 1830. V.P. 1832.

1845 JOHN CLARK, M.D., Staff Surgeon, 1st class; West Indies.

1848 JOHN CLARKE, M.D., Physician to the British Lying-in Hospital; Clifford-street, Bond-street.

1850 JOSIAH CLARKSON, Birmingham.

1835 JAMES CLAYTON, Percy-street, Bedford-square. C. 1850.

1842 OSCAR MOORE PASSEY CLAYTON, Percy-street, Bedford-square.

1853 JOSEPH T. CLOVER, Resident Medical Officer, University College Hospital.

1851 EDWARD COCK, Surgeon to Guy's Hospital; St. Thomas' s street, Southwark.

1850 DANIEL WHITAKER COHEN, M.D., Assistant-Physician to St. Thomas's Hospital; Cleveland-row, St. James's.

1835 *WILLIAM COLBORNE, Chippenham, Wiltshire.

1818 ROBERT COLE, F.L.S., Holybourne, Hampshire.

1828 JOHN CONOLLY, M.D. D.C.L., Hanwell, Middlesex.

1840 *WILLIAM ROBERT COOKE, Burford, Oxfordshire.

1820 BENJAMIN COOPER, Stamford.

1819 GEORGE COOPER, Brentford, Middlesex.

1841 GEORGE LEWIS COOPER, Surgeon to the Bloomsbury Dispensary; Woburn-place, Russell-square.

1843 WILLIAM WHITE COOPER, Senior Surgeon to the North London Eye Infirmary, to the Honorable Artillery Company, and Ophthalmic Surgeon to St. Mary's Hospital; Berkeley-square.

Elected

1841 HOLMES COOTE, *Secretary;* Demonstrator of Anatomy at St. Bartholomew's Hospital; Queen-square, Bloomsbury.

1835 GEORGE FORD COPLAND, Cheltenham.

1822 JAMES COPLAND, M.D. F.R.S., *President;* Consulting Physician to Queen Charlotte's Lying-in Hospital; Old Burlington-street. C. 1830. V.P. 1838.

1847 JOHN ROSE CORMACK, M.D., Putney, Surrey.

1839 *CHARLES CÆSAR CORSELLIS, M.D., Resident Physician to the Lunatic Asylum, Wakefield, Yorkshire.

1853 WILLIAM GILLETT CORY, M.D., Sutton, Surrey.

1847 RICHARD PAYNE COTTON, M.D., Assistant-Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges-street, Piccadilly.

1828 WILLIAM COULSON, Surgeon to the Magdalen Hospital, Consulting Surgeon to the City Lying-in Hospital, and Senior Surgeon to St. Mary's Hospital; Frederick's-place, Old Jewry. C. 1831. L. 1832. V.P. 1851.

1817 *SIR PHILIP CRAMPTON, Bart., F.R.S., Surgeon-General to the Forces in Ireland; Dublin.

1841 MERVYN ARCHDALL NOTT CRAWFORD, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Upper Berkeley-street, Portman-square. C. 1853.

1822 SIR ALEXANDER CRICHTON, M.D. F.R.S. and F.L.S., Physician in Ordinary to their Imperial Majesties the Emperor and Dowager Empress of all the Russias; the Grove, Sevenoaks, Kent. C. 1823.

1847 GEORGE CRITCHETT, Assistant-Surgeon to the London Hospital, and the Royal London Ophthalmic Hospital; Finsbury-square.

1837 JOHN FARRAR CROOKES, Russell-square.

1849 *WILLIAM EDWARD CROWFOOT, Beccles, Suffolk.

1851 JAMES CAMERON CUMMING, M.D., 1, Cadogan-place, Sloane-street.

1818 WILLIAM CUMING, M.D., Professor of Botany at the Glasgow Institution, and Surgeon to the Royal Infirmary at Glasgow.

1846 HENRY CURLING, Ramsgate, Kent.

Elected

1837 THOMAS BLIZARD CURLING, F. R. S., Surgeon to, and Lecturer on Surgery at, the London Hospital; New Broad-street, City. S. 1845. C. 1850.

1847 JOHN EDMUND CURREY, M.D., Lismore, Ireland.

1836 GEORGE CURSHAM, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, and to the Female Orphan Asylum; Savile-row, Regent-street. S. 1842. C. 1850.

1822 CHRISTOPHER JOHN CUSACK, Chateau d'Eu, France.

1852 THOMAS CUTLER, M.D., Physician to the Spa General Dispensary; Spa, Belgium.

1828 ADOLPHE DALMAS, M.D., Paris.

1851 NATHANIEL JOHN DAMPIER, Surgeon to the Farringdon General Dispensary and Lying-in Charity; Woburn-place, Russell-square.

1836 *JAMES STOCK DANIEL, Ramsgate.

1850 JOHN BAMPFYLDE DANIELL, M.D., Physician to the Royal Pimlico Dispensary; Grosvenor-street, Grosvenor-square.

1820 GEORGE DARLING, M.D., Russell-square. C. 1841.

1818 *SIR FRANCIS SACHEVEREL DARWIN, Knt., M.D., Breadsall Priory, near Derby.

1848 HENRY DAUBENY, 34A, York-street, Gloucester-place.

1846 FREDERICK DAVIES, Surgeon to the Northern Dispensary; Upper Gower-street, Bedford-square.

1818 *HENRY DAVIES, M.D., 6, Duchess-street, Portland-place. C. 1827. V.P. 1848.

1847 JOHN DAVIES, M.D., Physician to the Hertford Infirmary, and Visiting Physician to the County Gaol and Lunatic Asylum, Hertford.

1853 ROBERT COKE NASH DAVIES, Winchelsea, Sussex.

1852 WILLIAM DAVIES, M.D., Senior Physician to the United Hospital, Bath; Gay-street, Bath.

1852 JOHN HALL DAVIS, M.D., Russell-place, Fitzroy-square.

1820 THOMAS DAVIS, Spring-gardens. C. 1843.

1818 JAMES DAWSON, Liverpool.

1847 GEORGE EDWARD DAY, M.D. F.R.S., Chandos Professor of Medicine, St. Andrew's.

Elected

1841 CAMPBELL DE MORGAN, Surgeon to, and Lecturer on Physiology at, the Middlesex Hospital; Upper Seymour-street, Portman-square. S. 1851-2.

1846 *SAMUEL BEST DENTON, Ivy-lodge, Hornsea, East Riding, Yorkshire.

1844 ROBERT DICKSON, M.D., Hertford-street, May-fair.

1839 JAMES DIXON, *Librarian;* ~~Assistant Surgeon to St. Thomas's Hospital, and~~ Surgeon to the Royal London Ophthalmic Hospital; Green-street, Park-lane.

1845 JOHN DODD, Bryanston-street, Portman-square.

1846 JOHN DRUMMOND, Deputy Inspector of Fleets and Hospitals; Royal Naval Hospital, Chatham.

1843 THOMAS JONES DRURY, M.D., Physician to the Salop Infirmary; Shrewsbury.

1845 GEORGE DUFF, M.D., 53, Upper Seymour-street, Portman-square.

1845 EDWARD WILLSON DUFFIN, Langham-place, Portland-place.

1833 ROBERT DUNN, Norfolk-street, Strand. C. 1845.

1843 CHRISTOPHER MERCER DURRANT, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 HENRY SUMNER DYER, M.D., Bryanston-square.

1836 JAMES WILLIAM EARLE, Norwich.

1824 GEORGE EDWARDS.

1823 CHARLES CHANDLER EGERTON, Kendall-lodge, Epping.

1848 GEORGE VINER ELLIS, Professor of Anatomy in University College, London; 52, Albert-street, Regent's-park.

1835 WILLIAM ENGLAND, M.D., Wisbeach, Cambridgeshire.

1842 JOHN ERIC ERICHSEN, Professor of Surgery in University College, London, and Surgeon to University College Hospital; Welbeck-street, Cavendish-square.

1815 *GRIFFITH FRANCIS DORSETT EVANS, M.D., High-street, Bedford. C. 1838.

1836 GEORGE FABIAN EVANS, M.D., Physician to the General Hospital, Birmingham.

1845 WILLIAM JULIAN EVANS, M.D.

1841 SIR JAMES EYRE, M.D., Consulting Physician to St.George's and St. James's Dispensary; Brook-street, Grosvenor-square. C. 1851.

1844 ARTHUR FARRE, M.D. F.R.S., Professor of Midwifery in King's College, London; Hertford-street, May-fair.

1831 ROBERT FERGUSON, M.D., Physician-Accoucheur to the Queen, Physician to the Westminster Lying-in Hospital; Park-street, Grosvenor-square. C. 1839. V.P. 1847.

1841 WILLIAM FERGUSSON, F.R.S., Professor of Surgery in King's College, London; Surgeon to King's College Hospital, and to H.R.H. Prince Albert; George-street, Hanover-square. C. 1849.

1852 ALFRED GEORGE FIELD, 46, Great Marlborough-street.

1850 *FREDERICK FIELD, Birmingham.

1849 GEORGE TUPMAN FINCHAM, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital, and Physician to the Western Dispensary; 28, Chapel-street, Belgrave-square.

1836 JOHN WILLIAM FISHER, Surgeon-in-Chief to the Metropolitan Police Force; Grosvenor-gate. C. 1843.

1838 GEORGE LIONEL FITZMAURICE, Gloucester-place, Portman-square.

1842 THOMAS BELL ELCOCK FLETCHER, M.D., Physician to the General Hospital, Birmingham.

1841 SIR JOHN FORBES, M.D. F.R.S. D.C.L., Knt., Physician to her Majesty's Household; Old Burlington-street. C. 1852.

1848 JOHN GREGORY FORBES, Surgeon to the Western General Dispensary; Devonport-street, Hyde-Park.

1852 JOHN COOPER FORSTER, Southwell, Notts.

1817 *ROBERT THOMAS FORSTER, Southwell, Notts.

1820 THOMAS FORSTER, M.D., Hartfield-lodge, East Grinstead.

1816 JOHN W. FRANCIS, M.D., Professor of Materia Medica in the University of New York, U.S.

1841 JOHN CHRISTOPHER AUGUST. FRANZ, M.D., Royal German Spa, Brighton.

1843 PATRICK FRASER, M.D., Physician to the London Hospital; Guilford-street, Russell-square.

1836 JOHN GEORGE FRENCH, Surgeon to St. James's Infirmary; Great Marlborough-street, Regent-street. C. 1852-3.

1849 ROBERT TEMPLE FREERE, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Queen-street, May-fair.

Elected

1846 HENRY WILLIAM FULLER, M.D., Assistant-Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; Manchester-square.

1815 *GEORGE FREDERICK FURNIVAL, Egham, Surrey.

1851 GEORGE GASKOIN, Cambridge-terrace, Hyde-park.

1819 JOHN SAMUEL GASKOIN, Clarges-street, Piccadilly. C. 1836.

1819 HENRY GAULTER.

1848 JOHN GAY, Surgeon to the Royal Free Hospital; Finsbury-place, Finsbury-square.

1821 *RICHARD FRANCIS GEORGE, Surgeon to the Bath Hospital.

1851 STEPHEN JENNINGS GOODFELLOW, M.D., Physician to the Royal General Dispensary, and Assistant-Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Russell-square.

1818 JAMES ALEXANDER GORDON, M.D. F.R.S., Burford-lodge, Box-hill. C. 1828. V.P. 1829.

1851 PETER YEAMES GOWLLAND, Finsbury-square.

1844 JOHN GRANTHAM, Crayford, Kent.

1850 HENRY GRAY, F.R.S., Surgeon to the St. George's and St. James's Dispensary; Wilton-street, Grosvenor-place.

1846 GEORGE THOMPSON GREAM, M.D., 2, Upper Brook-street, Grosvenor-square.

1816 JOSEPH HENRY GREEN, F.R.S., Consulting Surgeon to St. Thomas's Hospital; Hadley, Middlesex. C. 1820. V.P. 1830.

1843 ROBERT GREENHALGH, Surgeon-Accoucheur to the Royal General Dispensary, St. Pancras; 11, Upper Woburn place, Russell-square.

1814 JOHN GROVE, M.D., Salisbury.

1852 JOHN GROVE, Wandsworth, Surrey.

1849 WILLIAM WITHEY GULL, M.D., Assistant-Physician to Guy's Hospital; Finsbury-square.

1837 JAMES MANBY GULLY, M.D., Holyrood-House, Great Malvern.

1819 SIR JOHN GUNNING, Knight, C.B., Inspector of Hospitals; Paris.

1842 CHARLES WILLIAM GARDINER GUTHRIE, Surgeon to the Westminster Hospital, and to the Westminster Ophthalmic Hospital; Pall Mall East.

Elected

1849 HAMMETT HAILEY, Newport Pagnell, Bucks.

1852 ROBERT JAMES HALE, M.D., 17, Westbourne Terrace, Hyde-park.

1842 *GEORGE HALL, M.D.

1845 JOHN HALL, M.D., Deputy Inspector-General of Hospitals; Cape of Good Hope.

1848 ALEXANDER HALLEY, M.D., Queen Anne-street, Cavendish-square.

1819 THOMAS HAMMERTON, Piccadilly. C. 1829.

1838 HENRY HANCOCK, Surgeon to the Charing-cross Hospital; Harley-street, Cavendish-square. C. 1851.

1849 *RICHARD JAMES HANSARD, Broad-street, Oxford.

1848 *GEORGE HARCOURT, M.D., Chertsey, Surrey.

1836 JOHN FOSSE HARDING, Mylne-street, Myddleton-square.

1843 THOMAS SUNDERLAND HARRISON, M.D. F.L.S., Garston-lodge, Somersetshire.

1846 JOHN HARRISON, the Court-yard, Albany.

1841 WILLIAM HARVEY, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity; Soho-square.

1853 ARTHUR HILL HASSALL, M.D., Physician to the Royal Free Hospital; 8, Bennett-street, St. James's.

1828 CÆSAR HENRY HAWKINS, President of the Royal College of Surgeons of England, Senior Surgeon to St. George's Hospital; Grosvenor-street, Grosvenor-square. C. 1830. V.P. 1838. T. 1841.

1838 CHARLES HAWKINS, Savile-row, Regent-street. C. 1846. S. 1850.

1848 THOMAS HAWKSLEY, M.D., George-street, Hanover-square.

1820 THOMAS EMERSON HEADLAM, M.D., Newcastle-upon-Tyne.

1848 *JAMES NEWTON HEALE, M.D., Physician to the Winchester County Hospital; Winchester.

1850 GEORGE HEATON, M.D., Boston, U.S.

1829 THOMAS HEBERDEN, M.D., Park-street, Grosvenor-square.

1844 JOHN HENNEN, M.D., Physician to the Western General Dispensary; Upper Southwick-street, Hyde-pk. L. 1848.

1848 MITCHELL HENRY, Assistant-Surgeon to the Middlesex Hospital; Harley-street, Cavendish-square.

Elected

1849 AMOS HENRIQUES, Upper Berkeley-street, Portman-square.

1821 VINCENT HERBERSKI, M.D., Professor of Medicine in the University of Wilna.

1843 PRESCOTT GARDNER HEWETT, Assistant-Surgeon to the St. George's Hospital, Lecturer on Anatomy at St. George's Hospital Medical School; Hertford-street, May-fair.

1841 *NATHANIEL HIGHMORE, Consulting-Surgeon to the Weymouth and Dorsetshire Eye Infirmary; Sherborne, Dorsetshire.

1814 *WILLIAM HILL, Wooton-under-Edge, Gloucestershire.

1842 WILLIAM AUGUSTUS HILLMAN, Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, Westminster Hospital; Argyll-street, Regent-street.

1841 JOHN HILTON, F.R.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; New Broad-street, City. C. 1851.

1848 MARTIN THOMAS HISCOX, M.D., Bath, Somersetshire.

1840 THOMAS HODGKIN, M.D., Bedford-square. C. 1842.

1813 JOSEPH HODGSON, F.R.S., Westbourne-terrace, Hyde-park. C. 1817. P. 1851.

1835 THOMAS HENRY HOLBERTON, Hampton, Middlesex.

1843 LUTHER HOLDEN, 54, Gower-street, Bedford-square.

1814 SIR HENRY HOLLAND, Bart., M.D. F.R.S., Physician to the Queen, and Physician in Ordinary to H.R.H. Prince Albert; Brook-street, Grosvenor-square. C. 1817. V.P. 1826.

1846 BARNARD WIGHT HOLT, Surgeon to the Westminster Hospital; Parliament-street, Westminster.

1846 CARSTEN H. HOLTHOUSE, Surgeon to the Public Dispensary, Lincoln's Inn; Assistant-Surgeon to, and Lecturer on Anatomy and Physiology at, the Westminster Hospital; 9, New Burlington-street.

1819 *JOHN HOWELL, M.D. F.R.S.E.; Clifton, Gloucestershire.

1828 *EDWARD HOWELL, M.D., Swansea, Glamorganshire.

1844 EDWIN HUMBY, Windsor-terrace, Maida-hill.

1822 ROBERT HUME, M.D. C.B., Inspector of Hospitals; Commissioner in Lunacy; Curzon-street, May-fair. V.P. 1836.

1840 HENRY HUNT, M.D., Brook-street, Hanover-square.

Elected

1842 CHRISTOPHER THOMAS AGRIPPA HUNTER, Downham, Norfolk.

1849 EDWARD LAW HUSSEY, Surgeon to the Radcliffe Infirmary, Oxford.

1820 WILLIAM HUTCHINSON, M.D.

1840 CHARLES HUTTON, M.D., Physician to the Royal Infirmary for Children; Assistant-Physician to the General Lying-in Hospital; Lowndes-street, Belgrave-square.

1848 GEORGE COCKBURN HYDE, 5, Halkin-street, Belgrave-sq.

1838 WILLIAM IFIL, M.D.

1847 WILLIAM EDMUND IMAGE, Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1826 WILLIAM INGRAM, Midhurst, Sussex.

1839 ALEXANDER RUSSELL JACKSON, M.D., Warley Barracks, Essex.

1845 *HENRY JACKSON, Surgeon to the Sheffield General Infirmary; St. James's Row, Sheffield.

1841 PAUL JACKSON, Bentinck-street, Manchester-square.

1847 THOMAS REYNOLDS JACKSON, Charles-street, St. James's.

1841 MAXIMILIAN MORITY JACOBOVICZ, M.D., Pesth.

1825 JOHN B. JAMES, M.D.

1847 *WILLIAM WITHALL JAMES, Exeter, Devonshire.

1844 SAMUEL JOHN JEAFFRESON, M.D., Leamington, Warwickshire.

1839 JULIUS JEFFREYS, F.R.S., Bath, Somersetshire.

1840 *GEORGE SAMUEL JENKS, M.D., Brighton.

1851 WILLIAM JENNER, M.D., Professor of Pathological Anatomy in University College, and Assistant-Physician to University College Hospital, Albany-street, Regent's park.

1848 ATHOL ARCHIBALD WOOD JOHNSON, Lecturer on Physiology at St. George's Hospital Medical School, and Surgeon to the Hospital for Sick Children; 37 Albemarle-street.

1851 EDMUND CHARLES JOHNSON, M.D., Savile-row, and Arlington-street, Piccadilly.

1821 SIR EDWARD JOHNSON, M.D., Weymouth, Dorsetshire.

1847 GEORGE JOHNSON, M.D., Assistant-Physician to King's College Hospital; Woburn-square.

Elected

1837 HENRY CHARLES JOHNSON, Surgeon to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; Savile-row, Regent-street. C. 1850.

1844 JOHN JOHNSTON, Old Burlington-street.

1844 HENRY BENCE JONES, M.D., F.R.S., Physician to St. George's Hospital; Grosvenor-street, Grosvenor-square.

1835 HENRY DERVICHE JONES, Soho-square.

1853 THOMAS WHARTON JONES, F.R.S., 35, George-street, Hanover-square.

1837 THOMAS WILLIAM JONES, M.D., Physician to the City Dispensary; Finsbury-pavement, Finsbury-square.

1829 *GEORGE CHARLES JULIUS, Richmond, Surrey.

1816 *GEORGE HERMANN KAUFFMANN, M.D., Hanover.

1815 ROBERT KEATE, Serjeant-Surgeon to the Queen, Surgeon to H.R.H. the Duchess of Gloucester; Hertford-street, May-fair. C. 1818. V.P. 1826.

1848 *DANIEL BURTON KENDELL, M.D., St. John's, Wakefield, Yorkshire.

1847 ALFRED KEYSER, Norfolk-crescent, Oxford-square.

1839 *DAVID KING, M.D., Eltham, Kent.

1851 JOHN ABERNETHY KINGDON, New Bank-buildings, City.

1836 PETER NUGENT KINGSTON, M.D., Physician to the Westminster Hospital; 8, Kensington-gate, Hyde-park. C. 1846.

1840 SAMUEL ARMSTRONG LANE, Lecturer on Anatomy; Surgeon to the Lock Hospital, and to St. Mary's Hospital; Grosvenor-place, Hyde-park. C. 1849.

1841 *CHARLES LASHMAR, M.D., Croydon, Surrey.

1816 G. E. LAWRENCE.

1809 WILLIAM LAWRENCE, F.R.S., Surgeon Extraordinary to the Queen; Surgeon to St. Bartholomew's Hospital, and to Bridewell and Bethlem Hospital; Lecturer on Surgery at St. Bartholomew's Hospital; Whitehall-place, Whitehall. S. 1813. V.P. 1818. C. 1820. T. 1821. P. 1831.

1840 THOMAS LAYCOCK, M.D., York.

1843 *JESSE LEACH, Heywood, near Bury, Lancashire.

1823 JOHN G. LEATH, M.D.

Elected

1822 JOHN JOSEPH LEDSAM, M.D.

1822 ROBERT LEE, M.D. F.R.S., Physician to the British Lying-in Hospital; Physician-Accoucheur to the St. Marylebone Infirmary; and Lecturer on Midwifery at St. George's Hospital; Savile-row, Regent-street. C. 1829. S. 1830. V.P. 1835.

1823 HENRY LEE, M.D., Keppel-street, Russell-square. C. 1837. S. 1839.

1843 HENRY LEE, Assistant-Surgeon to King's College Hospital, and Surgeon to the Lock Hospital; Dover-street, Piccadilly.

1851 GEORGE MACARTNEY LEESE, Gloucester-place, Portman-square.

1836 FREDERICK LEIGHTON, M.D., Franckfort-on-the-Maine.

1847 JOHN CHARLES WEAVER LEVER, M.D., Physician-Accoucheur to Guy's Hospital; Wellington-street, Southwark.

1847 SIR JOHN LIDDELL, M.D. F.R.S. C.B., Inspector of Hospitals; Royal Hospital, Greenwich.

1806 JOHN LIND, M.D.

1845 WILLIAM JOHN LITTLE, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 34, Brook-street, Grosvenor-square.

1819 ROBERT LLOYD, M.D.

1824 EUSEBIUS ARTHUR LLOYD, Surgeon to St. Bartholomew's and Christ's Hospitals; Bedford-row. S. 1827. V.P. 1838. C. 1843.

1820 J. G. LOCHER, M.C.D., Town Physician of Zurich.

1844 EDWARD FRANCIS LONSDALE, Surgeon to the Royal Orthopædic Hospital; Montague-street, Russell-square.

1824 CHARLES LOCOCK, M.D., First Physician-Accoucheur to the Queen, and Consulting Physician to the General Lying-in Hospital; Hertford-street, May-fair. C. 1826. V.P. 1841.

1852 CHARLES LODGE, M.D., Rye-lane, Peckham, Surrey.

1846 HENRY THOMAS LOMAX, Stafford.

1836 JOSEPH S. LÖWENFELD, M.D., Berbice.

1815 *PETER LUARD, M.D.

Elected

1852 JAMES LUKE, Senior-Surgeon to the London Hospital; Vice-President of the Royal College of Surgeons of England; Broad-street Buildings.

1846 WILLIAM M'EWEN, M.D., Surgeon to the Cheshire County Gaol, and House-Surgeon to the Chester General Infirmary; Newgate-street, Chester.

1814 SIR JAMES MACGRIGOR, Bart., M.D. K.C.B. K.T.S. LL.D. F.R.S. L. and E., Director-General of the Medical Department of the Army; Harley-street, Cavendish-square. C. 1820. V.P. 1815.

1823 GEORGE MACILWAIN, Consulting Surgeon to the Finsbury Dispensary: the Court-Yard, Albany. C. 1829. V.P. 1848.

1839 WILLIAM MACINTYRE, M.D., Harley-street, Cavendish-square. C. 1850.

1848 FREDERICK WILLIAM MACKENZIE, M.D., Chester-place, Hyde-park-square.

1818 WILLIAM MACKENZIE, Surgeon to the Eye Infirmary, Glasgow.

1822 RICHARD MACKINTOSH, M.D.

1844 DANIEL MACLACHLAN, M.D., Physician to the Royal Hospital, Chelsea, and Deputy Inspector-General of Hospitals; Royal Hospital, Chelsea.

1851 SAMUEL MACLEAN, Brook-street, Grosvenor-square.

1849 DUNCAN MACLACHLAN MACLURE, Harley-street, Cavendish-square.

1842 JOHN MACNAUGHT, M.D., Bedford-street, Liverpool.

1835 DANIEL CHAMBERS MACREIGHT, M.D., St. Hillier's, Jersey.

1837 ANDREW MELVILLE M'WHINNIE, Lecturer on Comparative Anatomy at St. Bartholomew's Hospital; Assistant-Surgeon to the London Hospital for Diseases of the Skin; Bridge-street, Blackfriars. C. 1851.

1848 WILLIAM ORLANDO MARKHAM, M.D., Assistant-Physician to St. Mary's Hospital; Clarges-street, Piccadilly.

1824 SIR HENRY MARSH, Bart., M.D., Dublin.

1838 THOMAS PARR MARSH, M.D. [redacted] the Salop Infirmary, Shrewsbury.

Elected

1851 JOHN MARSHALL, Assistant-Surgeon to University College Hospital, 10, George-street, Hanover-square.

1841 JAMES RANALD MARTIN, F.R.S., Lower Grosvenor-street, Grosvenor-square. C. 1853.

1819 *JOHN MASFEN, Surgeon to the County General Infirmary, and Fever Hospital, Stafford.

1849 GEORGE BELLASIS MASFEN, 78, Oxford-street, Manchester.

1853 WILLIAM EDWARD MASFEN, Stafford.

1818 J. P. MAUNOIR, Professor of Surgery at Geneva.

1837 THOMAS MAYO, M.D. F.R.S., Physician to the St. Marylebone Infirmary; Wimpole-street, Cavendish-square. S. 1841. C. 1847. V.P. 1851.

1839 RICHARD HENRY MEADE, Bradford, Yorkshire.

1819 *THOMAS MEDHURST, Hurstbourne Tarrant, Hampshire.

1837 SAMUEL WILLIAM JOHN MERRIMAN, M.D., Physician to the Royal Infirmary for Children; Consulting Physician to the Westminster General Dispensary; and Assistant-Physician to the West London Lying-in Institution; 3, Charles-street, Westbourne-terrace, Hyde-park.

1852 JAMES MERRYWEATHER, 57, Brook-street, Grosvenor-square.

1847 EDWARD MERYON, M.D., Clarges-street, Piccadilly.

1815 AUGUSTUS MEYER, M.D., St. Petersburgh.

1840 RICHARD MIDDLEMORE, Consulting-Surgeon to the Eye Infirmary, Birmingham.

1818 *PATRICK MILLER, M.D. F.R.S.E., Physician to the Devon and Exeter Hospitals, and to the Lunatic Asylum; Exeter, Devonshire.

1848 GAVIN MILROY, M.D., Fitzroy-square.

1852 JAMES MONRO, M.D., Surgeon-Major, Coldstream Guards; Vincent-square, Westminster.

1844 NATHANIEL MONTEFIORE, 4, Stanhope-street, May-fair.

1828 JOSEPH MOORE, M.D., Physician to the Royal Freemasons' Female Charity; Consulting Physician to Queen Charlotte's Lying-in Hospital; Savile-row, Regent-street. C. 1837.

1836 GEORGE MOORE, M.D., Hastings.

Elected

1848 CHARLES HEWITT MOORE, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 35, Montague-place, Russell-square.

1851 FREDERICK JOHN MOUAT, M.D., Professor of Medicine in the Medical College of Calcutta, and Secretary of the Council of Education in India; Calcutta.

1814 *GEORGE FREDERICK MUHRY, M.D., Hanover.

1847 SIMON MURCHISON, Steepleaston, near Woodstock, Oxon.

1841 EDWARD WILLIAM MURPHY, M.D., Professor of Midwifery in University College, London; Henrietta-street, Cavendish-square.

1845 THOMAS D. MUTTER, M.D., Professor of Surgery in Jefferson Medical College; Philadelphia.

1849 ROBERT NAIRNE, M.D., *Treasurer;* Physician to, and Lecturer on Medicine at, St. George's Hospital; Charles-street, Berkeley-square. C. 1848.

1835 THOMAS ANDREW NELSON, M.D., Nottingham-terrace, New-road.

1843 EDWARD NEWTON, Howland-street, Fitzroy-square.

1851 JAMES NICHOLS, Savile-row, Regent-street.

1816 THOMAS NIXON (Army).

1819 *GEORGE NORMAN, Surgeon to the United Hospital and Puerperal Charity; Bath.

1849 HENRY BURFORD NORMAN, Surgeon to the North London Eye Infirmary, and the St. Marylebone Dispensary; Duchess-street, Portland-place.

1845 HENRY NORRIS, South Petherton, Somerset.

1849 *ARTHUR NOVERRE, Great Stanmore, Middlesex.

1847 *WILLIAM EDWARD CHARLES NOURSE, 11, Orchard-street, Portman-square.

1843 WILLIAM O'CONNOR, M.D., George-street, Portman-square.

1847 THOMAS O'CONNOR, March, Cambridgeshire.

1846 FRANCIS ODLING, Devonshire-street, Portland-place.

1822 JAMES ADEY OGLE, M.D. F.R.S., Clinical and Aldrichian Professor of Medicine, Oxford; and Senior Physician to the Radcliffe Infirmary; Oxford.

1850 HENRY OLDHAM, M.D., Obstetric Physician to Guy's Hospital; Devonshire-square, Bishopsgate-street.

Elected

1842 WILLIAM PIERS ORMEROD.

1846 *EDWARD LATHAM ORMEROD, M.D., Physician to the Sussex County Hospital; Old Steyne, Brighton.

1847 WILLIAM EMANUEL PAGE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Curzon-street, May-fair.

1847 *WILLIAM BOUSFIELD PAGE, Surgeon to the Cumberland Infirmary; Carlisle.

1840 JAMES PAGET, F.R.S., Assistant-Surgeon to, and Lecturer on General and Morbid Anatomy and Physiology at, St. Bartholomew's Hospital; Henrietta-street, Cavendish-square. C. 1848.

1806 *ROBERT PALEY, M.D., Bishopston-grange, near Ripon, Yorkshire.

1836 S. W. LANGSTON PARKER, Surgeon to the Queen's Hospital; Birmingham.

1847 NICHOLAS PARKER, M.B., Assistant Physician to the London Hospital: Microscopical Demonstrator of Morbid Anatomy at the London Hospital School of Medicine; Finsbury-square.

1841 JOHN PARKIN, M.D., Paris.

1851 JAMES PART, 7, Camden-road Villas, Camden-town.

1828 RICHARD PARTRIDGE, F.R.S., Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; New-street, Spring-gardens. S. 1832. C. 1837. V.P. 1847.

1845 THOMAS BEVILL PEACOCK, M.D., Assistant-Physician to St. Thomas's Hospital; Finsbury-circus, Finsbury-sq.

1830 CHARLES P. PELECHIN, M.D., St. Petersburgh.

1819 JOHN PRYOR PEREGRINE, M.D., Jersey.

1839 THOMAS PEREGRINE, M.D. Half Moon-street, Piccadilly.

1844 WILLIAM VESALIUS PETTIGREW, M.D., Chester-street, Grosvenor-place.

1837 BENJAMIN PHILLIPS, F.R.S., *Vice-President;* Brentbridge House, Hendon, Middlesex. L. 1841. T. 1847.

1814 *EDWARD PHILLIPS, M.D., Physician to the County Hospital; Winchester, Hampshire.

1848 EDWARD PHILLIPS, M.D., Coventry, Warwickshire.

Elected

1852 Richard Phillips, 5, Winchester-place, Pentonville.

1846 Francis Richard Philp, M.D., Physician to St. Luke's Hospital; Colby-house, Kensington.

1851 *James Hollins Pickford, M.D., M.R.I.A., Brighton.

1851 John Picton, M.D.

1836 Isaac Pidduck, M.D., Physician to the Bloomsbury Dispensary; Montague-street, Russell-square.

1852 George Pilcher, Harley-street, Cavendish-square.

1852 Henry Pilleau, 11, Young-street, Kensington-square.

1841 Henry Alfred Pitman, M.D., *Librarian;* Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Montague-place, Russell-square.

1850 Alfred Poland, Assistant-Surgeon to Guy's Hospital, and to the Royal Ophthalmic Hospital; St. Thomas's-street, Southwark.

1845 George David Pollock, Surgeon to the North London Eye Infirmary; Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital Medical School; Grosvenor-street, Grosvenor-square.

1843 Charles Pope, M.D. M.A. F.L.S., Temple Cloud, near Bristol.

1846 Jephson Potter, M.D. F.L.S., Oxford-road, Manchester.

1840 Lewis Powell, John-street, Berkeley-square.

1842 James Powell, M.B. (Lond.), Guildford-street, Russell-square.

1851 Robert Francis Power, M.D., Bolton-street, Piccadilly.

1839 John Propert, New Cavendish-street, Portland-place.

1845 John Pyle, Surgeon to the North London Eye Infirmary; Oxford-terrace, Hyde-park.

1816 Sir William Pym, M.D., Inspector of Hospitals.

1830 Jones Quain, M.D., Paris.

1850 Richard Quain, M. D., Assistant-Physician to the Hospital for Consumption; Harley-street, Cavendish-square.

1835 Richard Quain, F.R.S., *Treasurer;* Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London; Cavendish-square. C. 1838. L. 1846. T. 1851.

Elected

1852 CHARLES BLAND RADCLIFFE, M.D., Assistant Physician to the Westminster Hospital; Henrietta-street, Cavendish-square.

1821 HENRY REEDER, M.D., Ridge House, Chipping, Sudbury.

1835 G. REGNOLI, Professor of Surgery in the University of Pisa.

1846 JAMES REID, M.D., Physician to the Infirmary of Gt. Giles and Bloomsbury; General Lying-in Hospital, &c.; Brook-street, Grosvenor-square.

1847 SAMUEL RICHARDS, M.D., Bedford-square.

1853 CHRISTOPHER THOMAS RICHARDSON, M.B., 16, Hinde-street, Manchester-square.

1829 SIR JOHN RICHARDSON, Knt. F.R.S. C.B., Surgeon to the Naval Hospital; Haslar Hospital, Gosport.

1849 *WILLIAM RICHARDSON, M.D., 9, Ephraim-road, Tunbridge Wells, Kent.

1843 JOSEPH RIDGE, M.D., Dorset-square.

1845 BENJAMIN RIDGE, M.D., Putney, Surrey.

1852 CHARLES RIDLEY, Charlotte-street, Bedford-square.

1852 JOHN ROBERTS, M.D., Physician to the Westminster General Dispensary; Bruton-street, Berkeley-square.

1829 *ARCHIBALD ROBERTSON, M.D. F.R.S. L. and E., Physician to the General Infirmary, Northampton.

1843 GEORGE ROBINSON, M.D., Newcastle-on-Tyne.

1851 RICHARD RADFORD ROBINSON, Camden-row, Camberwell, Surrey.

1843 WILLIAM RODEN, M.D. F.L.S., Kidderminster.

1835 GEORGE HAMILTON ROE, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Upper Brook-street, Grosvenor-square. C. 1841.

1836 ARNOLD ROGERS, Hanover-square.

1846 WILLIAM RICHARD ROGERS, M.D., Berners-street, Oxford-street.

1819 HENRY SHUCKBURGH ROOTS, M.D., Consulting-Physician to St. Thomas's Hospital; Russell-square. C. 1833. V.P. 1834.

1829 WILLIAM SUDLOW ROOTS, Kingston, Surrey.

1850 GEORGE ROPER, 180, Shoreditch.

1836 RICHARD ROSCOE, M.D., Twickenham, Middlesex.

Elected

1836 *Caleb Burrell Rose, Swaffham, Norfolk.

1850 Archibald Colquhoun Ross, M.D., Madeira.

1849 Charles Henry Felix Routh, M.D., 52, Montague-square.

1845 Henry Mortimer Rowdon, 27, Nottingham-place, York-gate, Regent's-park.

1841 Richard Rowland, M.D., Assistant-Physician to the Charing-cross Hospital; Woburn-place, Russell-square.

1834 Henry William Rumsey, Cheltenham.

1845 James Russell, M.D., Physician to the General Dispensary, Birmingham.

1851 Henry Hyde Salter, M.B., Mantague-street, Russell-sq.

1827 *Thomas Salter, F.L.S., Poole, Dorsetshire.

1844 *Thomas Bell Salter, M.D., F.L.S., Ryde, Isle of Wight.

1849 Hugh James Sanderson, Upper Berkeley-street, Portman-square.

1847 William Henry Octavius Sankey, M.D. London Fever Hospital, Liverpool-road, Islington.

1845 Edwin Saunders, Surgeon-Dentist to the Queen, and Lecturer on Diseases of the Teeth at St. Thomas's Hospital; George-street, Hanover-square.

1834 Ludwig V. Sauvan, M.D., Warsaw.

1840 Augustin Sayer, M.D., Upper Seymour-street, Portman-square.

1853 Maurice Schulhof, M.D., Suffolk-place, Pall Mall.

1824 Edward James Seymour, M.D. F.R.S., Charles-street, Berkeley-square. C. 1826. S. 1827. V.P. 1830.

1840 William Sharp, F.R.S. F.G.S. F.R.A.S., Rugby.

1837 William Sharpey, M.D. F.R.S. L. and E., Professor of Anatomy and Physiology in University College, London; Gloucester-crescent, Regent's-park. C. 1848.

1836 Alexander Shaw, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; Henrietta-street, Cavendish-square. C. 1842. S. 1843. V.P. 1851.

1848 *Edward James Shearman, M.D., Rotherham, Yorkshire.

1849 Francis Sibson, M.D. F.R.S., Physician to St. Mary's Hospital; Brook-street, Grosvenor-square.

1848 Edward Henry Sieveking, M.D., Assistant-Physician to St. Mary's Hospital; Bentinck-street, Manchester-square.

Elected

1839 THOMAS HOOKHAM SILVESTER, M.D., High-street, Clapham.

1842 JOHN SIMON, F.R.S., Lecturer on Pathology at St. Thomas's Hospital; Lancaster-place, Strand.

1821 CHARLES SKENE, M.D., Professor of Anatomy and Surgery; Marischal College, Aberdeen.

1827 GEORGE ROBERT SLENE, Bedford.

1824 FREDERIC CARPENTER SKEY, F.R.S., Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Northern Dispensary; Grosvenor-street, Grosvenor-square. C. 1828. L. 1829. V.P. 1841.

1838 HENRY SMITH, Senior Assistant-Surgeon to St. Mary's Hospital; and Lecturer on Surgery in the Medical School adjoining St. George's Hospital; Upper Seymour-street, Portman-square.

1835 JOHN GREGORY SMITH, Harewood, Yorkshire.

1843 ROBERT WILLIAM SMITH, M.D. M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; Dublin.

1852 CHARLES CASE SMITH, Senior-Surgeon to the Suffolk General Hospital; Bury St. Edmunds, Suffolk.

1845 WILLIAM SMITH, Park-street, Bristol.

1847 WILLIAM SMITH, M.D., Weymouth, Dorsetshire.

1850 WILLIAM TYLER SMITH, M.D., Physician-Accoucheur to St. Mary's Hospital; Upper Grosvenor-street, Grosvenor-square.

1843 JOHN SNOW, M.D., Sackville-street, Piccadilly.

1819 *GEORGE SNOWDEN, Ramsgate, Kent.

1851 JOHN SODEN, Surgeon to the Bath Hospital; Bath.

1816 *JOHN SMITH SODEN, New Sidney-place, Bath.

1830 SAMUEL SOLLY, F.R.S., Surgeon to St. Thomas's Hospital; St. Helen's Place, Bishopsgate-street. L. 1838. C. 1845. V.P. 1849.

1844 FREDERICK ROBERT SPACKMAN, M.B., Harpenden, St. Alban's.

1834 JAMES SPARK, Newcastle, Staffordshire.

1851 ROBERT JOHN SPITTA, M.B., Clapham, Surrey.

Elected

1843 *Stephen Spranger, Grantham, Lincolnshire.

1838 George James Squibb, Orchard-street, Portman-square.

1815 Edward Stanley, F.R.S., Surgeon to St. Bartholomew's Hospital; Brook-street, Grosvenor-square. C. 1821. S. 1824. V.P. 1827. T. 1832. P. 1843. C. 1852, 3.

1851 James Startin, Surgeon to the Hospital for Diseases of the Skin, and Lecturer on Cutaneous Disorders at that Institution; Savile Row, Regent-street.

1852 Shebard Freeman Statham, Assistant-Surgeon to University College Hospital; 43, Mortimer-street, Cavendish-square.

1852 John Edward Stephens, M.D., 9, Southwick Crescent, Hyde-park.

1842 Alexander Patrick Stewart, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; Grosvenor-street, Grosvenor-square.

1843 Robert Reeve Storks.

1844 John Soper Streeter, Harpur-street, Red Lion-square.

1847 William Allen Sumner, Surgeon to the Portland Town Free Dispensary; 25 Wellington Road, St. John's-wood.

1839 Alexander John Sutherland, M.D., F.R.S., Physician to St. Luke's Hospital; Richmond Terrace, Whitehall. C. 1850.

1842 James Syme, Professor of Clinical Surgery in the University of Edinburgh; Charlotte-square, Edinburgh.

1844 Richard William Tamplin, Surgeon to the Royal Orthopædic Hospital; Old Burlington-street.

1848 Thomas Hawkes Tanner, M.D., Physician to the Hospital for Women, Red Lion-square; Charlotte-street, Bedford-square.

1840 Thomas Tatum, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; George-st., Hanover-sq. C. 1852, 3.

1835 John Colley Taunton, Surgeon to the City of London Truss Society, and to the City Dispensary; Hatton-garden, Holborn. C. 1840.

1845 Thomas Taylor, Vere-street, Cavendish-square.

Elected

1852 Robert Taylor, M.D., 82, Guildford-street, Russell-square.

1845 *Evan Thomas, Manchester.

1839 Seth Thompson, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Lower Seymour-street, Portman-square. C. 1849. S. 1850.

1842 Theophilus Thompson, M.D. F.R.S., Physician to the Hospital for Consumption and Diseases of the Chest; Bedford-square.

1852 Henry Thompson, Surgeon to the St. Marylebone Dispensary; 16, Wimpole-street, Cavendish-square.

1835 Frederick Hale Thomson.

1819 John Thomson, M.D. F.L.S., Physician to the Finsbury Dispensary; Dalby-terrace, Islington. C. 1833. S. 1834. V.P. 1850.

1850 Robert Dundas Thomson, M.D., Professor of Chemistry, University of Glasgow.

1836 John Thurnam, M.D., Devizes, Wiltshire.

1848 Edward John Tilt, M.D., Physician to the Farringdon Dispensary; York-street, Portman-square.

1834 Robert Bentley Todd, M.D. F.R.S., Physician to King's College Hospital, Professor of Physiology and of General and Morbid Anatomy in King's College, London; Brook-street, Grosvenor-square. L. 1842. T. 1850.

1828 James Torrie, M.D., Aberdeen.

1843 Joseph Toynbee, F.R.S., Surgeon to the St. George's and St. James's Dispensary, and Aural Surgeon to St. Mary's Hospital; Savile-row, Regent-street.

1850 Samuel John Tracy, Surgeon-dentist to St. Bartholomew's and Christ's Hospitals; Finsbury-place, Finsbury-square.

1808 Benjamin Travers, F.R.S., Surgeon Extraordinary to the Queen, Surgeon in Ordinary to His Royal Highness Prince Albert; Green-street, Grosvenor-square. C. 1810. V.P. 1817. P. 1827.

1841 Matthew Truman, M.D., Norland-square, Notting-hill.

1835 John Cusson Turner, M.D., Brighton.

Elected

1845 THOMAS TURNER, Surgeon to the Royal Manchester Infirmary, and Lecturer on Anatomy; Mosley-street, Manchester.

1846 ALEXANDER URE, Surgeon to St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary, 18 Upper Seymour-street, Portman-square.

1819 BARNARD VAN OVEN, M.D., Consulting Surgeon to the Charity for Delivering Jewish Lying-in Women; 22, Manchester-square.

1806 BOYER VAUX, M.D.

1839 WILLIAM RANDALL VICKERS, Baker-street, Portman-square.

1810 JAMES VOSE.

1828 BENEDETTO VULPES, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

1841 ROBERT WADE, Surgeon to the Westminster General Dispensary; Dean-street, Soho.

1823 WILLIAM WAGNER, M.D., Berlin.

1820 THOMAS WALKER, M.D., Physician to the Forces; Morro Velhio, Brazil.

1852 WALTER HAYLE WALSHE, M.D., Professor of the Theory and Practice of Medicine in University College, and Physician to University College Hospital: 40, Queen-Anne-street, Cavendish-square.

1851 HENRY HAYNES WALTON, Assistant-Surgeon to St. Mary's Hospital; Brook-street, Hanover-square.

1852 DANIEL WANE, M.D., 20, Grafton-street, Berkeley-square.

1846 NATHANIEL WARD, Assistant-Surgeon to, and Demonstrator of Anatomy at, the London Hospital; Broad-street-buildings, City.

1845 THOMAS OGIER WARD, M.D., Leonard-place, Kensington.

1821 WILLIAM TILLEARD WARD, Duncannon House, Brighton.

1846 JAMES THOMAS WARE, Surgeon to the Finsbury Dispensary, and to the Convalescent Institution, Russell-square.

1811 JOHN WARE, Clifton, near Bristol.

1814 MARTIN WARE, Russell-square, *Vice-President*. C. 1844. T. 1846.

1816 *CHARLES BRUCE WARNER, Cirencester, Gloucestershire.

1829 ELIAS TAYLOR WARRY, Wimborne, Dorsetshire.

Elected

1837 THOMAS WATSON, M.D., Henrietta-street, Cavendish-square. C. 1840. V.P. 1845. C. 1852.

1847 *THOMAS WATSON, Holbeach, Lincolnshire.

1840 WILLIAM WOODHAM WEBB, Gislingham, near Thwaite, Suffolk.

1842 FREDERIC WEBER, M.D., Physician to the St. George's and St. James's Dispensary; Norfolk street, Park-lane.

1835 JOHN WEBSTER, M.D. F.R.S., Consulting Physician to the St. George's and St. James's Dispensary; Brook-street, Grosvenor-square. C. 1843.

1844 WILLIAM WEGG, M.D., Physician to the St. George's and St. James's Dispensary; Maddox-street, Hanover-square.

1816 SIR AUGUSTUS WEST, Knt., Deputy-Inspector of Hospitals to the Portuguese Forces; Paris.

1842 CHARLES WEST, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; and Physician to the Hospital for Sick Children; Wimpole-street, Cavendish-square.

1841 THOMAS WEST, M.D. F.L.S., Daventry.

1828 JOHN WHATLEY, M.D.

1849 JOHN WHITE, the Albany, Piccadilly.

1852 JOHN WIBLIN, 73, Morland-place, Southampton.

1840 JOSEPH WICKENDEN, Birmingham.

1824 *WILLIAM JOHN WICKHAM, Surgeon to the Winchester Hospital; Winchester.

1844 FREDERICK WILDBORE, High-street, Shoreditch.

1837 GEORGE AUGUSTUS FREDERICK WILKS, M.D., Temple-walk, Matlock, Derbyshire.

1840 CHARLES JAMES BLASIUS WILLIAMS, M.D., F.R.S., Upper Brook-street, Grosvenor-square. C. 1849.

1829 ROBERT WILLIS, M.D., Barnes, Surrey. L. 1838.

1839 ERASMUS WILSON, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; Henrietta-street, Cavendish-square.

1839 JAMES ARTHUR WILSON, M.D., Physician to St. George's Hospital; Dover-street, Piccadilly. C. 1846.

1831 WILLIAM JAMES WILSON, Surgeon to the Manchester Infirmary; Manchester.

1850 *ROBERT STANTON WISE, M.D., Banbury, Oxon.

Elected

1825 THOMAS ALEXANDER WISE, India.
1851 JOHN WOOD, Old Burlington-street.
1841 GEORGE LEIGHTON WOOD, Surgeon to the Bath Hospital; Queen-square, Bath.
1848 WILLIAM WOOD, M.D., Kensington House, Kensington.
1843 JOHN WARD WOODFALL, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.
1847 ROBERT WOOLLASTON, 62, Gloucester-terrace, Hyde-park.
1833 THOMAS WORMALD, Assistant-Surgeon to St. Bartholomew's Hospital; Bedford-row. C. 1839.
1842 WILLIAM COLLINS WORTHINGTON, Surgeon to the Infirmary, Lowestoft, Suffolk.
1848 EDWARD JOHN WRIGHT, Kennington-row, Kennington.

[It is particularly requested, that any change of Title or Residence may be communicated to the Secretaries before the 1st of August in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1841 WILLIAM THOMAS BRANDE, F.R.S. L. and E., Professor of Chemistry at the Royal Institution of Great Britain; Royal Mint, Tower-hill.

1835 SIR DAVID BREWSTER, K.H. LL.D. F.R.S. L. and E., &c., Cor. Mem. Institute of France, &c.; Kingussie.

1853 BENJAMIN COLLINS BRODIE, B.A., Oxford, F.R.S.; 13, Albert Road, Regent's-Park.

1841 ROBERT BROWN, D.C.L. F.R.S., President of the Linnean Society; British Museum.

1835 The Very Rev. WILLIAM BUCKLAND, D.D. F.R.S., Dean of Westminster.

1847 EDWIN CHADWICK, Commissioner of the Board of Health.

1835 MICHAEL FARADAY, D.C.L. F.R.S., Cor. Memb. Institute of France; Royal Institution.

1841 SIR JOHN FREDERICK WILLIAM HERSCHEL, BART., D.C.L. F.R.S., President of the Royal Astronomical Society; Somerset-House.

1835 SIR WILLIAM JACKSON HOOKER, LL.D. F.R.S. L. and E. Royal Botanic Garden, Kew.

1847 RICHARD OWEN, F.R.S., Cor. Memb. Institute of France; Hunterian Professor to, and Curator of the Museum of, the Royal College of Surgeons of England.

1835 The Rev. ADAM SEDGWICK, A.M. F.R.S. &c., Woodwardian Lecturer, Cambridge.

1841 The Rev. WILLIAM WHEWELL,, D.D. F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 G. ANDRAL, M.D. Professor in the Faculty of Medicine, Paris.

1835 CARL JOHAN ECKSTRÖM, K.P.S. and W., Physician to the King of Sweden, First Surgeon to the Seraphim Hospital, Stockholm.

1835 W. J. EDWARDS, M.D. F.R.S., Member of the Institute of France, Paris.

1841 CHRISTIAN GOTTFRIED EHRENBERG, Berlin.

1835 BARON A. DE HUMBOLDT, Member of the Institute of France, &c., Berlin.

1841 JAMES JACKSON, M.D., Professor of Medicine in the University of Cambridge, Boston, U.S.

1843 BARON JUSTUS LIEBIG, M.D. F.R.S., Professor of Chemistry in the University of Giessen, &c.

1841 P. C. A. LOUIS, M.D., Physician to the Hôtel-Dieu, Member of the Royal Academy of Medicine, &c., Paris.

1841 F. MAGENDIE, M.D., Member of the Institute; Physician to the Hospital of the Salpêtrière; Paris.

1847 PROFESSOR CARLO MATTEUCCI, University of Pisa.

1841 JOHANN MULLER, M.D., Professor of Anatomy and Physiology, and Director of the Royal Anatomical Museum, Berlin.

1835 J. C. OERSTED, M.D., Professor of Physics in the University of Copenhagen, &c. &c.

1835 PROFESSOR ORFILA, Dean of the Faculty, &c. &c., Paris. Obit 1853.

1841 BARTOLOMEO PANIZZA, M.D., Pavia.

1850 CARL ROKITANSKY, M.D., Curator of the Imperial Pathological Museum at the University of Vienna, &c. &c.

1853 VALENTINE MOTT, M.D., New-York.

1843 PHILIBERT JOSEPH ROUX, Member of the French Institute; Surgeon-in-Chief of the Hôtel-Dieu; Professor in the Faculty of Medicine, Paris.

1835 C. J. TIMMINCK, Director of the Museum of Natural History of the King of Holland, Amsterdam.

1835 FREDERICK TIEDEMANN, M.D., Professor of Anatomy and Physiology, Heidelberg.

1841 JOHN C. WARREN, M.D., Professor of Anatomy and Surgery in the University of Cambridge, Boston, U.S.

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ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state, that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers, which, on grounds of general merit, are thought worthy of being published in its Transactions.

A COMPARATIVE VIEW

OF SOME OF THE MORE IMPORTANT POINTS OF

THE PATHOLOGY OF

RHEUMATIC AND NON-RHEUMATIC PERICARDITIS,

DEDUCED FROM AN ANALYSIS OF CASES.

BY

EDWARD LATHAM ORMEROD, M.D.,

CAIUS COLLEGE, CAMBRIDGE;
FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; PHYSICIAN TO THE SUSSEX COUNTY HOSPITAL.

Received October 4th.—Read November 9th, 1852.

Pathologists are much indebted to the late Dr. Taylor for having satisfactorily shown[1] that acute rheumatism is not so exclusively the cause of pericarditis as had been before supposed; and for having called attention to the importance of granular disease of the kidney in this point of view. Dr. Taylor followed up his elaborate statistical inquiry by publishing in full the details of many of the cases on which these calculations rested. And the minuteness of the analysis is justified by the minute accuracy of the original records, as reported in the 'Lancet.'[2] But into the question of the nature of non-rheumatic pericarditis he has not entered so fully as into that of its frequency.

[1] Med.-Chir. Trans., vol. xxviii, p. 453. See also 'Medical Times,' vol. xxi, p. 482.

[2] Lancet, 1845, p. 46.

There need be no long waiting in the wards of a large London hospital to see what are the merits of a numerical question concerning either acute rheumatism or granular disease of the kidneys. A careful examination of my note-books, at the period of the publication of Dr. Taylor's researches, showed that, new as those conclusions were to me, they were generally consistent with the facts observed and recorded in ignorance of them. A few lines might serve to show how nearly my more limited experience on this question of comparative frequency agrees with that of Dr. Taylor. But the difference in the comparative nature and practical importance of the rheumatic and non-rheumatic forms requires a larger space for its illustration. In the belief that such an inquiry will not be wholly useless or devoid of interest, I have ventured to bring the subject under consideration in the following pages.

To avoid any misconception, it may be proper to state at the outset, that the word pericarditis is used in the following pages to denote recent inflammation of the pericardium. The analysis refers exclusively to cases of this nature. Not to reject the evidence from the traces of old pericarditis, I have not availed myself of it here, from the feeling that, on grounds of this kind, I have nothing new to urge.

The means of investigation is an analysis of the more or less complete records of 1410 cases observed under nearly similar circumstances, that is, in the wards of different hospitals. Of these cases a disproportionately large number are of thoracic, especially cardiac disease, and of fever. The ratio of cases of phthisis and of granular disease of the kidneys is also very high; though I believe that it fairly presents the relative frequency in hospital practice. Of these 1410 cases, 1249=88·59 per cent. were not cases of rheumatism; 161=11·41 per cent. were admitted on account of rheumatism at least deserving the name of sub-acute, or suffered it while under observation. Of the whole number, 85=6 per cent. had recent pericarditis, observed during life, or discovered after death, or both. These cases were thus distributed:—

24 =	1·92 per cent.	occurred among the	1249 non-rheumatic cases.
61 =	37·88 „	„	161 rheumatic cases.
85 =	6·		1410

The circumstances under which these cases were collected forbid the above numbers being taken to represent the liability to pericarditis, either in acute rheumatism, or in other diseases. But the interest attached to individual cases of pericarditis, which caused so disproportionately large a number of instances of this form of disease to be recorded, extended equally to the rheumatic and non-rheumatic class of cases: so that, though the average frequency of the occurrence under these different circumstances is unduly, yet I believe it is equally, raised.

As to the mode of arrangement which has been adopted: I have not at command cases enough of pericarditis, referable to each separate cause other than rheumatism, to allow me to deduce by analysis a summary of the characteristic effects (if such there be) of each of these in particular. It has seemed better, therefore, to group them, and to speak of rheumatic pericarditis rather as contrasted with this non-rheumatic group, than as being but one of many forms of a disease, all separately important, and having each their own distinctive characters. The only general subdivision of which the non-rheumatic cases seem to admit is, according as the pericarditis has appeared referable to local causes, or of more unequivocal constitutional origin.

Thus arranged, the features which are not marked strongly enough to stand alone are quite merged in the group. We cannot institute a running comparison between the detailed history of rheumatic pericarditis, for which there are sufficient and suitable materials, and the ill-assorted mass of facts which results from the accumulation of cases of all other forms of this affection. The comparison must therefore be limited to certain points, which the common bearing of all these cases shows to be important, and concurrently supplies the means for investigating.

The age of the 61 subjects of rheumatic pericarditis, here

examined, was about 21, on the average. And, where the numbers are large enough to place the conclusion out of the reach of the disturbing influence of single unusual cases, it does not seem that the difference of sex, or the alternative of death or recovery, involves any notable deviation from this age. The age was, however, slightly greater in the male patients, and in the favorable cases.

The mean age of the 24 subjects of non-rheumatic pericarditis was 42 years, the extremes being 7 and 63 years. All of these cases but one, a woman aged 24, dying of dysentery, were of the male sex.

The majority of the rheumatic subjects appear to have been generally healthy: certainly they were not a cachectic class. The non-rheumatic cases form a strong contrast in this respect. Even in the cases where the pericardial inflammation was engrafted on acute disease, the condition of the patients, previous to the accession of such primary disease, could not be designated as generally healthy.

As to the different causes of the pericarditis:—

Rheumatic . .	[1]61	cases	coincided with acute rheumatism.
Non-rheumatic of local origin	7	„	ensued on inflammation of the lungs or pleura.
	2	„	„ malignant disease of the pericardium.
	1	„	„ old cardiac disease.
Non-rheumatic of constitutional origin	[2]6	„	coincided with granular disease of the kidney.
	4	„	„ hæmorrhage or exhaustion.
	2	„	„ scarlatina and erysipelas respectively.
	[3]2	„	were inexplicable.
	85		

[1] Doubts may be expressed concerning the rheumatic nature of two of these cases. One of these doubtful cases may possibly have been suffering rather from remittent fever, the other from a mechanical injury.

[2] 5 certainly; 1 questionably.

[3] Of the two inexplicable cases, one was a rapidly fatal obscure case, of which no examination was allowed after death. The other was a case

Such is the general distribution of these cases. Their details, however, allow the investigation to be carried further into some particular points of the pathology of the two forms of the disease. And first, of the rheumatic class:—

The date of the accession of pericarditis was determined with tolerable accuracy in 33 of the above rheumatic cases. The mean of these observations gives the 10·5th day of the rheumatic attack as that on which the pericardial complication most commonly supervenes. In some other cases, pericarditis did not come on till 5, 6, 9, or 10 weeks from the beginning of the rheumatism. During this long period, however, the rheumatic attack was rather what might be called rheumatic disturbance of the constitution[1] than regular rheumatic fever. In these few late cases, the pericarditis, when it did come, was certainly very slight. But, as concerns the other cases, I am rather inclined to think that pericarditis supervening after the 10th day was more severe than when occurring at an earlier period.

The question, whether a first or a subsequent attack of rheumatism be more likely to be accompanied by pericarditis, is beyond the reach of hospital statistics. The circumstances which bring this class of rheumatic patients

of sudden fatal hæmorrhage into the pericardium, of which no explanation could be offered, save what a little patch of inflamed membrane near the base of the heart afforded. I may add that, but for the anomalous nature of the case which induced so very careful an examination, this morbid appearance might very easily have been overlooked, and certainly would not have been recorded.

On the rest of the non-rheumatic cases, there is only to remark here, that, in four of them, the pericardial affection might have been referred to more than one of the specified causes. Of these, one seemed referable to extension of inflammation rather than to erysipelas; another to renal rather than to old cardiac disease; the third rather to exhaustion, than to inflammation of a neighbouring part; and in the fourth, the old cardiac disease seemed in more active operation than the old renal disease.

[1] The rheumatic diathesis of Dr. Todd, on 'Gout and Rheumatism,' p. 109.

under observation, depend as much on personal considerations as on their disease. My note-books record more numerous admissions during a first, but proportionately more frequent instances of pericarditis during a second attack of acute articular rheumatism. The conclusion, however, among so many disturbing causes, whose influence I am unable to estimate, brings no conviction to my mind, and I forbear to urge it on such insufficient grounds.

Hospital statistics, in the same way, are unable to answer the question, whether pericarditis be more likely to occur in the severe or in the slighter cases of rheumatic fever. The rule cannot be absolutely and universally laid down on these grounds,[1] for hospital experience is limited, to an uncertain extent, to the severer cases of acute rheumatism. Within these limits, judging from the cases now under examination, it might be inferred that the degree of severity of the articular affection bears an inverse relation to that of the pericardial. Thus:—of the cases where the articular affection was noted as slight, in 53·3 per cent. the pericardial affection was severe; in 46·6 per cent. it was slight. Again, where the articular affection was noted as severe, the pericardial was severe in 42·8, and slight in 57·1 per cent. of the cases.

The inference from these figures, however, is somewhat weakened by the converse view of the case. Taking the degree of the severity of the pericarditis as the starting point: where this was slight, the same inverse ratio holds, the articular affection having been severe in 58·8, and slight in 41·2 per cent. But with severe pericarditis, the joints suffered severely in 73·7, and slightly in 26·3 per cent. of the cases under examination.

Without pressing the data to a more exact conclusion than their extent warrants, it may at least be safely inferred that the severity of the articular and pericardial affections bear no very close relationship to each other. Or, to speak

[1] Dr. Taylor, in his valuable paper already referred to, gives a summary of the discordant opinions held on this matter, drawn from such a partial view of the subject. (Op. cit., p. 523.)

from single extreme cases, whose practical warnings and encouragements alike are lost in an analysis, it may be said, on the one hand, that the most severe, even fatal, pericarditis, may come on in a case where clinical skill is taxed to the utmost to detect the articular affection. On the other hand, acute rheumatism, of whose severity no words short of agonizing and terrible can convey any adequate idea, by no means necessarily involves the addition of pericarditis to the other sources of distress.

In another point of view, namely, as to what is called the relation of metastasis from the joints to the heart, this series of cases supplies some facts worth consideration. There are 37 cases available for this inquiry. In one case pericarditis preceded the articular affection. In 6 it came on after the subsidence, and in 30 during the continuance of the affection of the joints. The antecedent case speaks for itself. As far as I can abstract my own judgment of the facts of the subsequent cases from an opinion averse to the theory of metastasis, I do not think that they bring any strong support to that theory. There appears nothing in these to justify any further statement than that the articular affection passed away; and then, after a variable time, pericarditis set in. The 30 cases, however, where disease existed at the same time in both the articulations and the pericardium, speak a less equivocal language; for in 4 only of these cases was any distinct relief to the joints observed on the accession of pericarditis.

As far as concerns the question of the likelihood of metastasis from the joints to the heart being the cause of pericarditis in rheumatism, the above cases furnish as strong negative evidence as could have been expected, viewed even in the most favorable light. It is quite unnecessary to reduce still further the value of the slight positive testimony which might be deduced from them in support of such a theory by a further analysis of such small results. Only it may be mentioned, that in such case the counter-irritant must be subtracted from the metastatic influence of pericarditis on the joints. And, in all fairness, against the few

cases where the articular has been relieved by the accession of pericardial inflammation, must be set those much more numerous ones where the recrudescence of the articular affection has brought with it pericarditis. And the converse.

It is a feature of rheumatic pericarditis that it allows these questions concerning it to be entertained. It is very characteristic of this form of the disease, that we can speak with such certainty of the date of its accession, which experience has led us to anticipate, and that we can weigh the severity of its local or constitutional symptoms. These considerations apply to this form exclusively. But, in pursuing the inquiry, there arise questions where the rheumatic and non-rheumatic forms mutually illustrate each other, and may be more advantageously studied together. The possible influence, for instance, of certain causes contingent on acute rheumatism, seems most fitly to come under examination with the influence of the same causes independent of rheumatism.

The chief questions involved in the history of non-rheumatic pericarditis of local origin, are the influence of pre-existent cardiac or pulmonary affections in inducing inflammation of the pericardium. Both these questions are of the utmost importance in relation to acute rheumatism also. And first as to pre-existent cardiac disease.

Of 60 of the cases of rheumatic pericarditis where the presence of endocarditis was sought for, in 6 only were no signs of endocardial lesion discovered. Of these 6 cases it is interesting to notice, that in 2 of them the pericarditis is described as severe. It is not to be inferred that acute endocarditis coincides, as a rule, in so large a proportion of cases with pericarditis. Some deduction must be made for cases of functional murmurs, and some for the existence of old valvular disease. But, though we cannot eliminate these sources of fallacy, the fact that the physical signs which might denote the effects of present or past endocarditis were found in nine tenths of these cases of rheumatic pericarditis is very important.

Was endocarditis, the precursor of pericarditis in so many of these cases, to be considered as its cause? There are no means of refuting such an hypothesis, on however slender grounds it may seem to rest. For pathology supplies us with no unequivocal instances of a tendency to acute endocarditis apart from a tendency to pericarditis also. Was pericarditis, where it preceded endocarditis, the cause of the secondary affection? Here the non-rheumatic cases of pericarditis supply the most important evidence against such an assumption. For as far as it is allowable to draw general conclusions from such small numbers, it appears that endocarditis is rarely met with in subjects of non-rheumatic pericarditis of the age and class of those now under examination. Indeed, in the absence of the cause of which they are both parallel effects, the frequency of their coincidence is not even as great as the neighbourhood of their seats respectively would have led one to anticipate.

The class of cases of non-rheumatic pericarditis, however, where a tendency to endocarditis does co-exist, is well worth attention. It includes infantile remittent fever, and the exanthemata of children. And though the following remarks[1] scarcely grow out of the cases under examination, where these diseases are but imperfectly represented, yet the seeming importance of the point, and its bearings on this part of our subject, may excuse the digression from the strict results of a tabular analysis.

[1] One of the cases marked as rheumatism in the series under examination may possibly have been remittent fever.

The cases under examination, do not, I believe, offer a fair idea of the frequency of pericarditis in cases of scarlatina; for hospital practice supplies comparatively few instances of scarlatina in young children, the age at which such an occurrence, according to recorded observations, is most likely. Among adults, of whom a large number are found suffering from scarlatina in St. Bartholomew's Hospital, pericarditis is certainly a very rare complication of this disease. As if, assuming its frequency in children, the tendency disappeared with advancing years.

On the subject of Pericarditis, as a complication of scarlatina in young subjects, besides the systematic works, see 'Lancet,' Dec. 14, 1839, p. 441; Mr. Snow.—'Med. Gazette,' vol. xxxv, p. 664; Dr. S. S. Alison.

There are at least two striking features which rheumatism with a tendency to heart affection has in common with these diseases. The most obvious is their more frequent occurrence in young subjects; and, where the occurrence of the disease itself is not restricted to early life, the loss of this tendency in more advanced age. The other less tangible feature is, that they all belong to the same class of blood-diseases so-called; to which indeed, by an extension of the term, the general pathological condition resulting from Bright's disease of the kidney might be referred. I say less tangible, for I cannot consider the vague term of blood-disease as more than a convenient form of expression in the absence of more accurate knowledge. We must wait the result of further inquiries like those of Dr. Garrod,[1] to give a definite meaning and permanent form to the expression, before we can safely rest any pathological theories upon it. For when we come to inquire more particularly into the nature of the deviations from the normal composition of the blood which characterise this class, we find no uniform condition[2] to which this particular effect, namely, the tendency to heart disease, might reasonably be ascribed. If we turn from the appreciable chemical changes to assumed changes in the vital properties of the blood, there indeed is a field for speculation; but who, in the present state of our knowledge, shall presume to build upon it?

Again, to return to our subject, as to the relation of pulmonary inflammation (using this as a general term) to pericarditis, the rheumatic and non-rheumatic forms mutually throw light on each other, and may be most advantageously considered together. In the 1410 cases which constitute the basis of the present inquiry, pulmonary inflammation—that is, pneumonia, pleuritis, or pleuro-pneumonia—was ascertained to exist, either by auscultation or dissection, 265 times. Of these—

[1] Med.-Chir. Trans., vol. xxxi, p. 83.

[2] Dr. Garrod; op. cit., p. 92.

117	had pneumonia, of which		19	had recent pericarditis.
86	„ pleurisy	„	6	„
62	„ pleuro-pneumonia		8	„
265			33 = 12·4 per. cent.	

As before, so many conditions may have exercised an influence on the observation and recording of these cases that it is not safe to take the figures as a general expression of the average frequency of the coincidence of pericardial and pulmonary inflammation. But, inquiring into the order of sequence and connection of the two local affections in the particular cases, we obtain the following results:—In 15 cases the pericardial preceded, in 8 it followed, the pulmonary inflammation. In five cases the date of accession of the two affections coincided, and in the remaining 5 the dates could not be determined accurately.

These results, however, require a little further arrangement before they can be made available for any general inferences; for the rheumatic and the non-rheumatic cases stand in strong contrast to each other. Of the 15 preceding cases 14 were rheumatic; while of the 8 subsequent cases all but 1 belonged to the other class.

Turning first to the relations of pulmonary and pericardial inflammation in the rheumatic class:—Observation shows that, though pericardial inflammation commonly precedes, yet that it sometimes, though rarely, follows pulmonary inflammation; and, more frequently, that pulmonary inflammation comes on without the intervention of pericarditis or endocarditis at all. There are thus no grounds for considering pulmonary inflammation as a consequence of pericarditis rather than as a conjoined effect of their common cause—acute rheumatism.

But, whatever the relation between the two affections, their coincidence has more than a mere abstract pathological interest. The coincidence is very frequent, and very important. It is very frequent; for in the 61 rheumatic cases now under examination, pulmonary inflammation

occurred 24 times—that is, in 39·3 per cent. of all the cases. Of these, 4 had pleurisy, of which 2 died; 17 had pneumonia, of which 4 died; and 3 had what may most properly be called pleuro-pneumonia, all of which recovered. It should be mentioned, by the way, that in some cases the disease began as bronchitis; but it ran on so rapidly to pneumonia that it seems most correct to designate it as such. And it is very important; for the question of life or death in cases of rheumatic pericarditis turns more often on the affection of the lungs than on that of the heart. Though it be true that rheumatism may induce pulmonary inflammation without the intervention of pericarditis, yet it seems as if this intervention were necessary to the manifestation of the pulmonary inflammation in all its severity.[1]

The non-rheumatic class tell quite a different story. Here pulmonary inflammation has apparently a distinct influence in inducing pericarditis. And this influence, as might have been expected, is most evident in cases of pleurisy. The clinical history, indeed, which in cases of rheumatism is commonly so precise, as to the date of the accession of pericarditis, is very deficient on this point in the non-rheumatic class. But, as far as anatomy goes, we may infer with certainty (and wherever clinical observation has anything to offer, it quite bears out the conclusion) that the pericarditis was subsequent to, and probably contingent on, the pulmonary inflammation.

To express the legitimate conclusions from the above considerations in a few words, pericarditis, in rheumatic cases, cannot be considered as a frequent result of pul-

[1] Considering the frequency and importance of the coincidence of rheumatic pericarditis and pulmonary inflammation, it is remarkable that it should have attracted so little attention, even of those whose detailed clinical observations show that they were quite familiar with it. I can refer only to Dr. Latham ('Lectures on Diseases of the Heart,' vol. i, p. 159,) for an express description of it. I would add my testimony, in which all who are familiar with the disease will agree, that his picture is not over drawn.

monary inflammation. The ultimate issue of the case, however, in death or recovery, is very much influenced by such a coincidence, which, moreover, is far from uncommon. Pericarditis, in non-rheumatic cases, not unfrequently may be referred to such a cause. But the coincidence is comparatively unimportant, the cardiac complication being rather a measure of the severity of the original disease than itself any serious addition to it. To this point, however, there will be occasion hereafter to return.

With these instances of propagation of inflammation to the pericardium from neighbouring structures, the occurrence of pericarditis in connection with malignant disease of this part (noticed in the preceding table), deserves a passing notice; for malignant disease of the pericardium, like malignant disease of the brain, seems to have an especial tendency to induce pericarditis about the period of the fatal termination, though the inflammation has not such a direct influence in bringing about this termination here as in the brain. The comparative rarity of this class of cases, however, and their hopeless nature, destroy whatever interest they might otherwise have possessed here in respect of this particular tendency.

It remains to inquire, in the same way, into the pathology of that class of cases where pericarditis depends on some general cause other than rheumatism. Besides the exanthematous fevers, which have already been cursorily noticed, the assumed causes under which the cases seem to group themselves are hæmorrhage, or exhaustion, and Bright's disease of the kidney. The effects of these causes, however, do not admit of being examined in the same way as those of the subdivision just now under consideration. There is little to say of either of them but what may more conveniently be expressed in a general summary of the characters of non-rheumatic pericarditis.

Hæmorrhage, or exhaustion, is mentioned among the less frequent causes. Though its independent influence appear small numerically expressed, there is reason to believe that, in connection with other causes, its influence in this respect

is very great;[1] for it may be doubted whether pericarditis occurring at the close of a disease be not more properly referable to the general effects of that disease on the constitution than immediately and specifically to the disease itself in many cases.

It would be very desirable to institute an exact comparison between Bright's disease of the kidney and acute rheumatism in respect to their tendencies to induce inflammation of the pericardium. The circumstances, however,

[1] In saying this, I would not be understood, as implying in any degree that the judicious employment of depletion in acute rheumatism has a tendency to induce pericarditis. It should require very strong evidence on the abstract point to deter any one from pursuing the plan dictated by common experience—that is, from using depletion, like other remedial means, according as the symptoms may indicate. And this strong evidence we certainly do not possess. Facts and opinions have not been collected and stated on this matter with the impartiality which alone could give us confidence in a conclusion at variance with ordinary experience. We must, indeed, receive with caution the recorded results of any plans of treatment pursued with such enthusiasm, as is the employment of depletion, by M. Bouillaud, in the treatment of acute rheumatism. Judged by the records of the practice of other physicians, the results of this more active treatment appear to be less favorable than those of ordinary practice. But the ill-effects of its excessive employment give no grounds for inferring that depletion is not applicable at all in these cases. We run into the opposite extreme, when by one sweeping clause we refer to the effects of depletion, what M. Bouillaud urges, as evidence of its having been necessary, and we hazard the well-being of our patients by such a hasty inference.

Of the 61 rheumatic cases under examination, blood was drawn by cupping or venesection in 7 cases before inflammation of the pericardium set in. It was drawn for the relief of the present symptoms, and more especially on account of present endocarditis. Now, obviously, the assumption that pericarditis resulted from depletion in these cases, however unjustifiable, yet cannot be refuted. The only answer that can be offered is the fact, that depletion has been employed on the same indications in a much larger number of cases of acute rheumatism, where yet pericarditis has not followed. The cases where depletion might in a similar way be said to have prevented rheumatic pericarditis, are at least three times as many as those where it might be said, as above, to have caused this complication; judging at least from my own observations.

under which these two diseases occur, and our sources of information concerning them, respectively, are so very different that they hardly admit of being put in comparison. Acute rheumatism is a disease, for instance, of short duration, with well-marked symptoms and well-known tendencies. Bright's disease of the kidney, from the present point of view, is in these respects its exact opposite. Our knowledge of the cardiac complications of acute rheumatism is mostly clinical; of those of Bright's disease mostly anatomical. The data for the statistics of acute rheumatism within hospital practice are tolerably distinct; but how often do we overlook Bright's disease in the living, and how we need some criterion, exact and of ready application, of its presence after death![1]

But though the data are wanting for an exact history of renal as compared with the other forms of pericarditis, yet, on the practical bearings of the subject, this class of cases is most instructive. Let us look at them in this point of view, grouping the renal with the other cases of non-rheumatic pericarditis. Though composed of such various elements, yet, to clinical observation, in its general features, negative as well as positive, the group is quite a natural one.

Turning, then, from the more particular examination of each form to a consideration of the general characters of the class, the negative features are those which come most strongly before us; for, looking at the clinical history of the disease, as compared with the rheumatic form, there appears to be quite a blank on those points where the features of rheumatic pericarditis are usually best marked; for there are no symptoms to mark its setting in, so severe as to make us forget the original disease in its complication, as there are in rheumatism. There is no fear of our insensibly acquiring impressions of a metastasis of disease from other parts to the pericardium, such as we need a

[1] Frerichs ('Die Bright'sche Nieren-krankheit,' s. 168,) thinks that the recognition of the fibrinous casts of the urinary tubes will, with the more general use of the microscope, supply this desideratum.

careful reference to our note-books to correct. The very existence of the disease was recognised during life, whether by symptoms or by physical signs, in scarcely more than half the cases.

What a contrast with the rheumatic cases, where pain is so general and often so severe a symptom! There, of 61 cases, in 1 only were the physical signs unobserved, and this not from the heart not being examined, but from the part of the membrane affected lying posteriorly out of the reach of auscultation; while in 11 out of these 24 non-rheumatic cases no symptoms led to the heart being examined, and the existing disease, in a large proportion of them at least, remained undetected during life.

But if the symptoms and physical signs fail us here, the opportunities for informing ourselves of the amount and anatomical characters of the disease by dissection are more than proportionately increased. Of the 24 cases under examination 22 = 91·6 per cent. died. Again, what a contrast to rheumatic pericarditis, where the average[1] mortality, in this series of cases, is 18 per cent.

These facts deserve a very careful examination; for on their correct interpretation depends the answer to be given to the two most important practical questions which can be asked concerning any acute disease—namely, as to the amount of distress and the danger to life which it involves.

To be so very frequently found after death, non-rheumatic pericarditis should be a disease of exceeding frequency, unless there be some peculiar liability to its occurrence about this period. Now, it is true that pericarditis, leaving its traces in white spots, is a much more common disease than the most diligent observer would suspect from clinical experience unaided by dissection. But there are no sufficient grounds for claiming these white spots, the evidence of old partial pericarditis, as the effects of non-rheumatic pericarditis exclusively.[2] We are not always

[1] An average, I believe, much raised by the number of fatal cases which my office necessarily brought under my notice.

[2] Dr. Taylor's inquiries led him to the conclusion that within the range

stumbling on general non-rheumatic pericarditis during life as we should be did its clinical bear the same ratio to its anatomical srequency as does that of the rheumatic form. The interpretation most agreeable to observation is that which makes the more frequent occurrence about the period of death a characteristic of non-rheumatic pericarditis.

Is it the cause of death? The mortality, on such an assumption, is so very great that one naturally turns to the symptoms observed during life for an explanation of this. Here, as we have already seen, is a blank; but the negative fact that in no more than 13 of these 24 cases was the existence of pericarditis ascertained during life, is full of meaning. For diseases, speaking generally, prove fatal by their local or their constitutional effects. Of the first:—comparing the ordinarily slight amount of local changes in this form with the ordinarily large amount in the rheumatic form (which yet rarely proves fatal in this way), we may reject this explanation of the mortality in non-rheumatic pericarditis as insufficient. Then, as to the constitutional effects, there is no specific fatal tendency apart from the visible rational symptoms of non-rheumatic pericarditis. The 13 cases where the disease was recognised and watched during life are very explicit on this point. Speaking generally, on their evidence, where non-rheumatic pericarditis was watched day by day, judged by its symptoms, it was a slight disease. Its presence or absence seemed almost without influence on the patient's general condition; and no idea of any specific depressing influence, apart from the more determinate symptoms, could reasonably be entertained: nor, where the disease was not recognised during life, does a careful review of the symptoms which were recorded in ignorance of the existence of pericarditis bring into notice any indications of important

of rheumatism, Bright's disease of the kidneys, and inflammation of neighbouring parts, the causes of the "white spots" are to be found (Med.-Chir. Trans., vol. xxviii, pp. 467, 474). There are, however, no sufficient data to guide us in distributing the particular cases under these several heads.

constitutional sympathy with the local disease. The pericarditis was overlooked, because, in fact, it had no symptoms.

Dissection, while it displays, at the same time explains the oversight. It shows us changes slight in degree, and, commonly, of recent origin. Neither the constitutional indications of morbid action, nor the organic results of such action, are of considerable amount. Dissection and clinical observation agree in the same conclusion, that the disease accompanies rather than causes death; that it does not destroy life, but ensues because the patients are already dying.[1]

The contrast of what is observed in rheumatic pericarditis may serve to point and confirm this conclusion. With all the severe pain and distress, with all the extensive local changes, in itself, and apart from its complications, rheumatic pericarditis is very rarely the cause of death. It would be unreasonable to infer that non-rheumatic inflammation of the same part, a disease with few or no symptoms, whether local or general, simple or specific, and with trivial local changes, could be so much more fatal. It remains open to inquiry how far the frequency of rheumatic pericarditis has been over-rated in comparison with the non-rheumatic form. But the practical importance of this last in individual cases sinks in the exact proportion that we admit the greater frequency of its occurrence.

Before leaving this part of the subject, it is necessary to call attention to some of the details of the cases which form exceptions to the general results of the analysis which I have

[1] Pericarditis was found in 13 of 292 dissections of cases of Bright's disease (=4·4 per cent.) recorded by different authors, and analysed by Frerichs ('Die Bright'sche Nieren-krankheit,' s. 120). Agreeing fully with the author of this excellent monograph, as to the obscurity of the pericardial complications of Bright's disease of the kidneys, I cannot assent to his statement (s. 131), that pericarditis (under these circumstances) is, as a rule, rapidly fatal by its interference with the action of the heart. Such an inference is not justified by my own, and I think not by Dr. Taylor's observations.

thus far followed. Generally speaking, non-rheumatic pericarditis might seem to be a mild disease, or rather an unimportant complication accompanying death, analogous to inflammation of other serous membranes, which is liable to occur under similar circumstances. The less striking instances of deviation from this general average, such as pericarditis incident on local mechanical injury supplies, it is needless to recall. The more marked cases, where the disease was accompanied by characteristic symptoms, and was a cause of distress, or even of death, are two in number:

One of these was a man suffering from mania, and who had attempted to commit suicide. Shortly before his death, becoming quieter and more lucid, he complained of pain in his chest, when, on examination, the physical signs of pericarditis were recognised. On dissection, the pericardium displayed intense vascular injection, with abundant inflammatory exudation of coagulable lymph and serum. It was matter of deep regret that the peculiar circumstances of the case should have prevented such active disease from being recognised at an earlier period, when that knowledge might have been made available for directing remedial measures.

The other case seeming to require more particular notice is that of a painter who had hand-drop on both sides, from which he had suffered for many years. He had pain on the right side of the head, which increased so as to seem to require the abstraction of blood by cupping. But no relief followed. He sank rapidly, and during the last day of his life was affected with convulsive movements resembling those of chorea, limited nearly, but not exclusively, to the right side. The body was examined at a period when decomposition was so far advanced as to render it difficult to recognise some of the slighter morbid changes. The pericardium, however, was distinctly seen to be coated with a thin layer of coagulable lymph. This layer was thicker in some places than in others, forming bands connecting the two opposite serous surfaces. In one space, over an old

"white patch," there was no new adventitious deposit. In the right hemisphere of the brain, near its lower surface, at the junction of the middle and posterior lobes, was a cavity of about a quarter of an inch in diameter, filled with an olive-coloured fluid. Its walls were lined with a ragged, dark-brownish membrane. The kidneys were small, tough, and granular; some urine found in the bladder was albuminous. In connection with the hand-drop it should be noticed, that the right *extensor communis digitorum* had its muscular substance replaced by ordinary adipose tissue.

I would here, in concluding my own observations, almost offer an apology for the dry and uninteresting manner in which I have presented a subject which more than any other engages the daily interest of the clinical student. But pericarditis has been drawn too often and too well, for me to have anything new to add to clinical sketches of this kind. My object has been rather to call attention to, and investigate some of the less prominent parts of the subject. It remains only briefly in conclusion to see how far these results agree with those of the published cases of Dr. Taylor, who made the subject of non-rheumatic pericarditis so peculiarly his own.

The mean age of Dr. Taylor's non-rheumatic cases was 38·43, that of his rheumatic was 20·52 years. Of the 16 non-rheumatic cases whose sex is named, only 5 were females. The connection with granular disease of the kidney was more frequent than in my cases, being displayed in 10, or perhaps 12, of these 17 cases. Pulmonary inflammation was noted in 5 cases. Altogether, in the coincidence in the age, the predominance of the male sex, the cachectic nature of the subjects generally, and the general identity of the more important reputed causes, the agreement between Dr. Taylor's non-rheumatic cases and my own is sufficiently close to assure me that they both belong to the same class.

The ratio of the mortality does not admit of comparison, Dr. Taylor having selected fatal cases only. But as to the general characters of the disease, all that has been deduced from my own analysis might have been as faithfully deduced

from Dr. Taylor's more elaborate descriptions. The resemblance of the general features of the two series is indeed very striking, more particularly as to the obscurity of the non-rheumatic form. Of Dr. Taylor's 17 cases, in 6 only was the disease recognised during life. In three or four cases the existence of old adhesions limiting the disease to the posterior part explained how, though sought for, it had remained undetected. But in the remaining cases the existence of the disease was not betrayed during life by its symptoms; it was not sought for, and was as completely overlooked as in many of the cases which came under my own observation.

Wanting that personal knowledge of the cases which is essential for reducing their facts to figures, I would not enter into a more detailed analysis, but rest on the general conclusions which obviously arise from them. They were fully as obscure as those which came under my own notice.[1] One arises from the perusal of those elaborate clinical reports, with a conviction that non-rheumatic pericarditis is more within the province of the anatomist than of the physician. These cases, like those already analysed, mark it as a disease with few or no symptoms, its physical signs recognised more often by a chance discovery than on the suggestions of the disease, and its morbid changes small in amount and apparently inactive. In these too, as in those which have come under my own observation, where an opportunity has occurred of watching the disease for some time previous to death, it has been apparently without effect on the general symptoms, its presence or absence being determined by the ear alone. And still, in these, its connection with the fatal termination has appeared to be that of a coincidence rather than of a cause.

[1] Dr. Lalor describing an epidemic of continued fever where pericarditis was a frequent complication, notices that one of the most unfavorable circumstances connected with it was its extreme obscurity. ('Dublin Journal,' vol. xiii, n. s., p. 114.)

ON THE DEVELOPMENT

OF

TORULÆ IN THE URINE,

AND ON

THE RELATION OF THESE FUNGI TO ALBUMINOUS AND SACCHARINE URINE.

BY

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PART I.

BEFORE proceeding to offer any remarks of my own on the subject of the present communication, I propose to place before the Society the opinions and observations in relation to torulæ in urine, entertained and recorded by others up to this time.

"Diabetic urine," writes Dr. Griffith,[1] "if left in a warm place, becomes covered with a frothy white layer, as if its surface had been sprinkled with flour. This is quite characteristic, and when once seen cannot be mistaken. This white froth is composed of a number of minute vegetable organisations, which have been denominated *torulæ;* they occur in all fermenting liquids, and their growth is by some considered, in relation to fermentation, in the light of cause and effect. They are figured in pl. ii, fig. 35; their development is very interesting. When first formed they are very minute spherical globules, composed of two coats, and filled with a liquid containing in suspension a number

[1] Practical Manual, pp. 49, 50.

of extremly minute granules; the globules enlarge, rise to the surface, and form the white scum. Some of the internal granules also enlarge, and become distinct nuclei. These continue expanding, the primary globule becomes elongated, and one of the enlarged nuclei bursts through the envelopes of the maternal cell and appears as a bud; this enlarges, others increase in the same manner. As the globules enlarge they become elongated, finally forming long, slender, jointed vegetables, as in pl. ii, fig. 35. These contain several nuclei, which are ready to bud out in the same manner as their parents have originally done. They seem to increase in two distinct ways: one is the budding process above mentioned, the other is the division of the parent cell. It is first divided by the increase of two, three, or more nuclei into as many separate parts. It then becomes contracted opposite the spaces between the continuous extremities of the internal young cells, finally forming distinct and independent plants, capable of further propagation in a similar manner."

Under the head of Torulæ in Diabetic Urine, Dr. G. O. Rees states,[1] "This fungoid vegetable growth, which is delineated on the plate, fig. 15, is characteristic of the existence of fermentation, and its presence may be regarded as a very correct microscopic test of the presence of sugar."

Dr. Golding Bird[2] gives the following account of "Torulæ" in saccharine urine:—"It is well known that in all saccharine fluids undergoing the alcoholic fermentation, minute confervoid, or fungoid vegetations, called torulæ, appear, and pass through certain definite stages of development. There is, indeed, considerable reason to believe that these vegetations bear to fermentation the relation of cause and effect. The arguments lately advanced by Professor Liebig, in opposition to this opinion, do not, to my mind, afford a satisfactory answer to the observations previously made on this subject.

"When urine contains but very small portions of sugar,

[1] Analysis of the Blood and Urine, 2d edit., p. 217.

[2] Urinary Deposits, 2d edit., p. 289.

too little even to affect its specific gravity materially, or to cause it to assume a diabetic character, certain phenomena are developed connected with the production of the vegetation of the genus torulæ or saccharomyces, which will at once point out the presence of sugar. These indications are of very great value, as a saccharine condition of the urine is not uncommon in dyspepsia and some other affections, and is, of course, of the highest importance in directing our treatment.

"When saccharine urine is left in a warm place, a scum soon forms on its surface, as if a little flour had been dusted upon it. This consists of minute oval bodies which soon enlarge from the development of minute granules visible in their interior. These continue expanding, and dilate the oval vesicle containing them into a tubular form; soon afterwards the internal granules become larger and transparent, and project from the exterior of the parent vesicle-like buds. The whole then resembles a jointed fungoid or confervoid growth, which ultimately breaks up, and a copious deposit of oval vesicles or spores fall to the bottom. All these stages of development, fig. 46, require but a few hours for their completion. If the deposited spores be placed in weak syrup, they rapidly germinate, and exciting fermentation, produce a new crop of torulæ. During the growth of the torulæ, bubbles of carbonic acid gas are evolved, and the urine at length acquires a vinous odour, sometimes accompanied by an odour of butyric acid. There are two kinds of urine which may be mistaken for saccharine, by the occurrence of a kind of fermentation not unlike that of fluids really containing sugar. I refer to the form of viscous fermentation which occurs in urine, and ending in the appearance of much ropy mucus. This has occurred to me repeatedly in specimens of urine containing cystine, the odour evolved being, however, disagreeable and sulphurous, quite distinct from the vinous odour of the alcoholic fermentation. Somewhat similar phenomena are occasionally presented by the urine of persons exhausted in health from scrofulous or syphilitic cachexia."

"Torulæ," observes Dr. Bence Jones,[1] "are by no means diagnostic of saccharine urine; but though they form very soon and very plentifully in diabetic urine, yet they may be constantly found in urine which contains no trace of sugar; and though they may lead you to look for sugar, they must never lead you to assert that sugar is certainly present in the urine in which they occur."

In his 10th Lecture on Albuminous Urine,[2] Dr. Jones also makes the following remarks:—

"There is a peculiar microscopic appearance in acid albuminous fluids to which M. Andral has directed attention. In the 'Annales de Chemie,' vol. lxxxiii, p. 885, there is a paper on the development of the penicilium glaucum, under the influence of acidification, in the albuminous fluids of health and disease, by MM. Andral and Gavarret. Serum of the blood diluted with twice its volume of water, and acidified by dilute sulphuric acid, usually, in twelve hours, gave vesicles, which elongate rapidly, forming a long, branching, jointed vegetable, of which drawings are given in the different stages of its development; albumen and acid are necessary for its growth: if, therefore, this vegetation is met with in the urine, we may immediately conclude that albumen exists in solution, and heat and nitric acid will certainly confirm the truth of your opinion."

The above observations comprise nearly all the information contained in the writings of English authors in reference to torulæ in the urine.

It appears then, from these extracts, that Drs. Griffith, G. O. Rees, and G. Bird, on the one side, regard the development of fungi in urine as affording a very valuable and decided test of the presence of sugar, the latter observer even considering it to be so delicate as to be capable of detecting such small portions of sugar as are too little even to affect the specific gravity of the urine materially, or to cause it to assume a diabetic character.

On the other hand, Dr. Jones states, that torulæ are by

[1] On Animal Chemistry, p. 121. [2] Loc. cit., pp 109-10.

no means diagnostic of saccharine urine, although they form very soon and very plentifully in diabetic urine. In making this statement, however, Dr. Jones does not adduce the reasons which have led him to adopt this view; the opinion, however, as we shall see hereafter, follows as a necessary consequence from the inquiries of MM. Andral and Gavarret, on the development of penicilium glaucum in acid albuminous fluids.

Dr. Jones likewise states that, "If this vegetation is met with in the urine, we may immediately conclude that albumen exists in solution, and that heat and nitric acid will certainly confirm the truth of your opinion."

There is therefore a very considerable discrepancy of opinion as to the value of the torulæ test as an indication of sugar in the urine.

I will now proceed to record the results of my own observations on the Development of Torulæ in Urine.

I set aside, in the first place, at different periods, a considerable number of samples of non-saccharine urine of all kinds; some of these were acid, others alkaline, a few contained albumen, but the majority were non-albuminous. The changes which ensued in the several samples were observed and registered from day to day. In a large proportion of the samples torulæ quickly became developed, while in others they did not appear at all; they presented many distinct appearances and conditions of development, all of which, after a time, however, were ascertained to belong to, and to be characteristic of, one and the same species of fungus, of which the following is a description. Three distinct stages in the development of this plant may be recognised, each of which we shall describe separately under the heads of sporules, thallus, and aërial fructification.

Sporules.—Of these two kinds exist.

The *first* make their appearance in urine at an early period, usually in the course of a few hours, the precise time is determined, however, by the nature of the urine and the temperature of the weather; they are first visible as innumerable minute vesicles or cells, of a perfectly globular

form, reflecting, when seen with an object-glass of one fourth of an inch focus, bright centres and dark outlines, and presenting a tolerably uniform size; when viewed, however, with a glass of one eighth of an inch focus, the shaded outlines nearly disappear, and the sporules are then observed to present considerable differences of size, from the $\frac{1}{6778}$th of an inch in diameter to the $\frac{1}{11222}$d of an inch; the larger all include a vesicular nucleus, sometimes placed in the centre of each sporule, at others it is eccentric; but the smaller sporules are not nucleated, and resemble in size and appearance the nuclei of the larger sporules; the cavities of all are occupied by a fluid containing granules. (Pl. 1, fig. 1.)

In a short space of time, generally in a few hours, the sporules multiply to such an extent as to form, first, distinct circular patches and afterwards a continuous scum on the surface of the urine, as contained in a bottle or glass. In this scum the sporules are not heaped up over each other, but form a delicate stratum, constituted of a single layer of sporules, which, while they evidently repel the water, yet adhere to each other. Sometimes the patches, although small, cease to grow; in other cases they extend, until they touch each other, and from being circular become angular from mutual compression, the several patches yet remaining distinct; very frequently, as soon as they touch each other, they run together and form a continuous stratum, as noticed above: this may remain, as indeed it often does, without undergoing further change, or it may become wrinkled, or thrown up in a waved manner; both these appearances arise from the extension of the single layer of sporules, which having covered the entire surface of the urine, and being unable to spread itself out further, becomes variously folded or plaited. In general, the patches, whether small or large, are thin, delicate, transparent, and film-like; but in some cases they appear dry and white, resembling flour; upon what this difference depends I am not clear, but I believe it is connected with the amount of phosphates present. It is in this latter state that the surface of the urine presents the powdery aspect considered by so many

observers as characteristic of saccharine urine. The smaller patches are usually, but not always, composed of the rounded sporules above described; sometimes the sporules have lost their spherical form, and this is almost constantly the case in the larger patches, and elongating slightly, become oval. (Pl. I, fig. 2.)

The elongation of the sporules is not constant, for sometimes the development of the fungus ceases with the formation of the globular sporules. This change of form occurs when the condition of the urine is such as to favour the growth of the fungus; under such condition the sporules quickly extend themselves, and become three or four times longer than broad, when they resemble short threads of nearly equal diameter (Pl. I, fig. 3), having rounded extremities; after a time, the sporules becoming still further elongated, pass into separate filaments, which consist of cells placed end to end, and all enclosed in a common transparent membrane; the threads are more or less curved, and increase in length sometimes by the extension of both extremities, but usually only of one, which is distinguished by its smaller diameter.

From this condition, which is frequently attained on the second or third day, the fungus passes into the higher state of *thallus*. (Pl. IV, fig. 1.)

The *second* kind of sporules, which for the sake of distinction I shall call *vesicles*, are many times larger than the ordinary sporules; their surface is frequently hirsute, like the pollen granules of the compositæ; they are globular, and from each proceed one, two, or three buds or shoots, which gradually extend into filaments, at first simple and afterwards branched, thus forming, as the sporules did in the previous case, the thallus.

Now while the ordinary sporules during growth are merged entirely into and lost in the filaments, the vesicles remain as prominent swellings or enlargements on the threads, not unfrequently increasing in size with the growth of the threads themselves. When the vesicles give origin to but a single filament, they are seen as terminal inflations; when to two

or more, they of course are situated in the midst of the ramifications which have emanated from them. (Pl. 1, fig. 4.)

The number of vesicles present varies greatly in different cases: in general, the ordinary sporules alone are met with; in others a few vesicles only occur mixed up with the common sporules; in others, the number of vesicles has been considerable; and again in a few rare cases, I have detected vesicles only in a state of germination.

Of the sporules and vesicles we shall shortly have to speak again.

Now in some urines the growth of the fungus goes no further than to produce the sporules and vesicles; at this point, and even at any stage, all development not unfrequently ceases. The cause of this singular circumstance will be explained hereafter.

We will now pass on to the description of the thallus.

Thallus.—The thallus, then, it appears, takes its origin either in the sporules or vesicles. The filaments or threads, at first simple, which proceed from these, afterwards become branched, and the myriads of threads developed interlace together. It is therefore a compound structure, made up of innumerable perfectly distinct plants, which are held together simply by the interlacement of the filaments.

Like the sporules, it forms a layer upon the surface of the urine often of considerable thickness, several days being usually required for its complete formation. The growth of the thallus takes place principally from the extremities of the filaments; these mostly lie the deepest in the fluid, and it is near the extremities also that the branchings are most numerous, and therefore best seen.

The filaments forming the thallus are comparable to the roots of higher plants, and they extend themselves for some distance through the fluid in which they are developed in search of the nourishment by which the fungus is sustained.

The cavities of the jointed and branched filaments, like those of the vesicles and sporules, are filled with granular and vesicular material.

Now the thallus is met with in urine in two states,—it either forms patches on the surface, or one continuous stratum, these states depending upon the number and distribution of the sporules which precede it. As is the case with the sporules, the development of the thallus may be arrested at any stage of its growth.

When this happens, it soon breaks up and dies; before the breaking-up of the threads occurs, however, I have frequently observed the granular and vesicular contents of the filaments to collect into little rounded or oval masses, which escaping through the common investing sheath of the threads, become so many sporules. When in a mass of thallus, some filaments are seen transparent and destitute of contents, while others contain little rounded or oval bodies; and when a large number of sporules are lying about intermixed with the threads, we know that this elimination of sporules has occurred.

Every perfect fungus developed in a fluid consists of two parts, an aquatic and an aërial. The thallus represents the aquatic portion, and the filaments of which it is formed readily imbibe the fluid in which they are immersed; on the other hand, the stems and sporules which form the aërial portion of the fungus, repel the water and manifest an affinity for the air.

The last stage, then, in the development of the fungus, is that of aërial fructification:

Aërial Fructification.—After the lapse of a still further time, a mouldiness appears on the surface of the already-formed thallus. This follows exactly the distribution of the thallus itself; if it be in patches, then the mouldiness will appear only in places on the surface of the urine; but if the thallus form a continuous stratum, the mould or mildew will do the same.

The mould or aërial fructification presents the following structural peculiarities:—

If the surface of the thallus be carefully examined sometime previous to the appearance of the mould or fructification, a number of short upright stems or threads will be observed.

Each vertical stem having attained a certain height, divides into one or two branches, each of which becomes subdivided into several other very short and slightly moniliform branches,—thus a tuft or head is formed; at the extremity of the several branches rows or strings of circular bodies appear; these, on the slightest movement, become detached from the head, and fall either on the thallus or into the water (Pl. IV, fig. 2); now these circular bodies are identical with the sporules first described, and each represents a separate plant. It is in these sporules that the glaucous green colour so characteristic of this fungus in its perfect state of development is located; the colour varies, however, greatly in different cases; sometimes the patches or stratum of the fungus possess scarcely a tinge of green, at others they are not in the least green, but of a fawn colour; lastly, in some cases in which the spore-bearing heads are not formed at all, the vertical threads, upon which when present they are supported, become considerably elongated, and then the patches resemble pieces of white wool.

Such is a short sketch of the development of this fungus in its different stages; it is to be observed, however, that the several conditions described do not always keep separate from each other; thus, frequently, the sporules and thallus coexist, and in some cases we find sporules, thallus, and fructification, all more or less mixed up together; in certain urines, successive generations even of sporules may be seen passing through the several phases of their development.

It has already been stated that the growth of this plant, from a cause to be mentioned presently, is frequently arrested at any one stage of its development; but this is not all, for soon afterwards it begins to decay, and finally disappears from the urine; the only trace of its presence remaining is a deposit of sporules, circular in form, but irregular in size, and situated at the bottom of the urine. After the plant has once attained its full development, however, many days must elapse before its total destruction and disappearance.

We will now conclude this description by a few remarks on the propagation of this fungus.

Its perpetuation appears to be provided for in several ways.

First.—By the sporules thrown or pinched off, as it were, from the reproductive tufts, and which therefore appear to be nothing more than definite and minute portions of the mother plant, each being endowed with independent vitality and capability of reproduction.

Second.—By the generation of secondary sporules within the cavities of the primary or first-formed sporules.

Third.—By the granular and vesicular matter which occupies the cavities of the filaments and cells forming the thallus; in these cavities circular bodies resembling somewhat sporules, but much smaller, may be seen, and it is probable that it is by their means that the species is perpetuated in those cases, of very frequent occurrence, in which the plant has been destroyed before attaining its full development.

Fourth.—By vesicles; these I regard as sporangia, analogous to the vesicular bodies, met with in the tribe of algæ, as parent-cells in fact, containing a number of germs.

The vesicles or sporangia are not confined to this one species, but are frequently to be met with in many other fungi, some belonging even to distinct genera.

Two other facts connected with the development of this fungus yet remain to be mentioned.

The first is, that it will develope itself with nearly similar facility, and in the same quantity, in urine passed directly into a new bottle and immediately corked, as in urine exposed to the air.

The second is, that sporules and even filaments may be detected in some urines almost immediately after they have been passed; from this it becomes probable that the development had commenced even in the bladder itself.

We have now to enter upon the consideration of the conditions necessary to the development of this fungus. The first step which I took with a view to determine what these conditions are, was to put aside in bottles, all corked,

a variety of urines, fixing the dates to each, and noting from time to time the changes which ensued; the results of this proceeding are given in the following table:—

1st SAMPLE:—*Aggravated Dyspepsia.* Urine very acid, non-albuminous, passed 24th August; on the 28th inst., a scum of circular sporules appeared; on the 2d of September, there was much of the fungus fully developed, with a few spherical sporules; on the 5th inst., the fungus was in the same state.

2d SAMPLE:—*Chlorosis*, with œdematous feet. Urine acid, passed on 17th of August; on the 20th inst., a scum of spherical sporules became visible; on the 2d and 8th of September, the sporules were in the same state.

3d SAMPLE:—*Dyspepsia.* Urine alkaline, non-albuminous, passed on the 22d of August; on the 24th inst., no sporules; on the 2d of September, no sporules.

4th SAMPLE:—*Disease of Liver*, Anasarca. Urine albuminous, alkaline, passed on the 19th of August; on the 23d inst., no trace of fungus; on the 2d of September, still no fungus.

5th SAMPLE:—*Dyspepsia.* Urine somewhat, but not strongly acid, non-albuminous, passed on the 17th of August; on the 20th inst., a few oval sporules; on the 2d of September, a scum of spherical sporules; on the 8th inst., sporules in same condition.

6th SAMPLE:—*Disease of Kidneys*, Anasarca. Urine albuminous, decidedly acid, passed on the 23d of August; on the 27th inst., a scum of elongated sporules and filaments; on the 2d of September, a thick and continuous stratum of the fungus fully developed.

7th SAMPLE:—*Disease of Kidneys.* Urine albuminous, feebly acid, passed on the 17th of August, examined on the same day, sporules with a few short filaments were visible; on the 20th inst., a few sporules, circular; on the 2d of September, sporules in the same state.

8th SAMPLE:—*Articular Rheumatism.* Urine somewhat acid, non-albuminous, passed on the 18th of August; on

the 20th inst., an incipient scum of spherical sporules; on the 2d of September, a very dense film of oval sporules; on the 8th inst., sporules in same condition.

9th SAMPLE:—*Chorea.* Urine passed on the 28th of August, not very acid, non-albuminous; on the 2d of September, a dense scum of spherical sporules; on the 14th inst., scum on surface of urine all gone, a few sporules irregular in size, at the bottom of the bottle.

10th SAMPLE:—*Phthisis.* Urine decidedly acid, slightly albuminous, passed on the 2d of September; on the 8th inst., spherical sporules abundant, with much fungus in a state of perfect fructification.

11th SAMPLE:—*Dyspepsia.* Urine neutral, non-albuminous, passed on the 2d of September; on the 8th inst., no sporules.

12th SAMPLE:—*Fever.* Urine feebly acid, non-albuminous, passed on the 19th of September; on the 20th inst., a few spherical sporules; on the 28th inst., the same; on the 2d of October, no increase in the number of sporules.

13th SAMPLE:—*Inflammation of Kidney.* Urine non-albuminous, acid, passed on the 21st of August; on the 26th inst., a scum of spherical sporules; on the 2d of Sept., sporules much elongated; on the 8th inst., fungus in same state.

14th SAMPLE:—*Disease of Kidneys,* Anasarca. Urine neutral; albuminous, passed on the 16th of August; on the 20th inst., no fungus; on the 2d of September, none.

15th SAMPLE:—*Typhus.* Urine alkaline, non-albuminous, passed on the 24th of August; on the 30th inst., no fungus; on the 2d of September, none.

16th SAMPLE:—*Fever.* Urine feebly acid, non-albuminous, passed on the 20th of August; on the 23d inst., powdery patches of circular sporules; on the 2d of Sept., a few sporules only.

17th SAMPLE:—*Aggravated Dyspepsia.* Urine somewhat acid, non-albuminous, passed on the 16th of August; on the 20th inst., a scum of spherical sporules; on the 2d of

September, a dense film of sporules; on the 8th inst., sporules in same state.

18th SAMPLE:—*Congestion of Liver.* Urine decidedly acid, non-albuminous, passed on the 21st of August; on the 26th inst., a scarcely perceptible scum of spherical sporules; on the 2d of September, sporules still circular; on the 8th inst., a patch or two of thallus.

19th SAMPLE:—*Disease of Brain.* Urine alkaline, non-albuminous, passed on the 21st of August; on the 24th inst., no sporules visible; on the 2d of September, none.

20th SAMPLE:—*Dyspepsia.* Urine alkaline, non-albuminous, passed on the 17th of August; on the 20th inst., no fungus; on the 2d of September, none.

21st SAMPLE:—*Fever.* Urine alkaline, non-albuminous, passed on the 28th of August; on the 30th inst., no fungus; on the 2d of September, none.

22d SAMPLE:—*Typhus.* Urine alkaline, non-albuminous, passed on the 19th of August; on the 23d inst., no fungus; on the 2d of September, none.

23d SAMPLE:—Urine acid, non-albuminous, passed on the 16th of August, at night; on the morning of the 18th inst., sporules appeared, some oval, others elongated; on the 20th inst., a dense scum of oval sporules and filaments; on the 2d of September, fungus in same state; on the 8th inst., still in the same condition.

24th SAMPLE:—*Phthisis.* Urine very acid, non-albuminous, passed on the 21st August; on the 2d of September, a scum of oval sporules; on the 8th inst., fungus fully developed.

25th SAMPLE:—Urine strongly acid, non-albuminous, passed on the 28th of August; on the 30th inst., an abundance of spherical sporules; on the 8th of September, a continuous scum of fully-developed fungus.

26th SAMPLE:—Urine acid, non-albuminous, passed on the 17th of August; on the 22d inst., spherical and oval sporules; on the 2d of September, a dense scum of circular sporules; on the 8th inst., sporules in the same state.

27th SAMPLE:—Urine acid, non-albuminous, passed on

the 19th of August; on the 22d inst., a thick scum of spherical sporules; on the 2d of September, sporules in the same state; on the 8th inst., sporules less numerous, many having sunk to the bottom.

28th SAMPLE :—*Typhus*. Urine feebly acid, non-albuminous, passed on the 18th of August; on the 20th inst., a few somewhat oval sporules; on the 2d of September, sporules in the same state.

29th SAMPLE :—Urine strongly acid, non-albuminous, passed on the 4th of September; on the 8th inst., circular sporules, with patches of fully-developed fungus.

30th SAMPLE :—Urine strongly acid, non-albuminous, passed on the 4th of September; on the 8th inst., sporules and filaments of fungus; on the 14th inst., fungus in perfect fructification.

31st SAMPLE :—*Phthisis*. Urine acid, non-albuminous, passed on the 30th of August; on the 8th of September, a scum of spherical sporules; on the 12th inst., no further development of the fungus.

32d SAMPLE :—Urine acid, non-albuminous, passed on the 30th of August; on the 8th of September, a scum of spherical sporules; on the 14th inst., sporules in the same state.

From an analysis of the above table it appears therefore—

1st. That the fungus was developed in twenty-four out of the thirty-two urines submitted to examination.

2d. That in thirteen samples the development did not proceed beyond the sporule-stage.

3d. That in two it was arrested when in the condition of thallus.

4th. That in the remaining nine urines, it attained its perfect state, viz., that of aerial fructification.

5th. That those urines in which the fungus made its appearance, were invariably more or less acid, the degree of development varying with the acidity.

6th. That those urines in which it failed to make its appearance, were either neutral or alkaline, or though acid when passed, very quickly became first neutral and then alkaline.

7th. That the fungus appeared alike in albuminous and non-albuminous urines, provided these were sufficiently acid.

The frequency of the presence of this fungus in the urine is thus clearly established. One of the conditions necessary to its development, as we have seen, is an acid state of the urine; the degree of acidity and the length of time during which the urine remains acid, regulating to a considerable extent the growth of the plant. When the acidity is great, and of some days' duration, other causes being favorable, the fungus is enabled to pass through all the stages of its development, and to reach the state of mould or perfect fructification; when, on the other hand, the acidity is but feeble, the growth proceeds but slowly, and ceases entirely at whatever stage it may happen to have attained on the passing of the urine from an acid to an alkaline condition, and which, as appears from the Table above given, very frequently occurs when the fungus is still in the first stage of its development, that of circular sporules.

As is also shown by the Table, for some days after the urine has ceased to be acid and has become alkaline, the fungus does not appear to undergo any material alteration, but at length it begins to decay, and finally disappears.

The condition of development of this fungus in any urine is therefore, to some extent, an indication of the degree of acidity once possessed by that urine; it must be remembered, however, that although this plant is never developed in alkaline urine, it is yet sometimes present in it, the urine having been in the first instance acid, and having become alkaline subsequently.

We have now to seek for other conditions necessary to the development of this fungus.

When it is remembered that fungi contain nitrogen in their composition, and when their constant association with dead or diseased organic matter is called to mind, the idea that the presence of the species under consideration in the urine is closely connected with the animal matter contained in that fluid will at once appear as highly probable.

Now, animal matter, and even albumen, as in mucus and epithelium, are constantly present in the urine in greater or less amount, the albumen being contained in the epithelial scales and mucous cells; and hence this fluid ordinarily supplies another of the conditions requisite for the growth of this fungus.

With the view to ascertain whether the notion just referred to was correct, I procured a number of urines. I divided each sample into two portions, one was carefully filtered so as to remove at least part of the animal matter, the other was allowed to remain just as it was passed; the whole of the samples were set aside in corked bottles, and examined from time to time. The results obtained by this proceeding were as follow:—

1st Sample:—Urine passed on the 13th of September; on the 20th inst. there was a very decided scum of sporules upon the surface of the unfiltered urine, but none upon the filtered; and on the 22d inst., still no appearance of fungus upon the surface of the latter.

2d Sample:—Urine passed on the 13th of September; on the 17th inst. a thick scum of sporules upon the unfiltered, but none upon the filtered urine; on the 22d inst. fungi on both, but the layer thickest on the unfiltered portion.

3d Sample:—Urine voided on the 9th of September; on the 14th inst. a scum upon the unfiltered, but none upon the filtered urine; and on the 17th inst. the scum was upon both, but much less thick upon the filtered portion.

4th Sample:—Urine voided on the 9th of September; on the 17th inst. no scum upon the filtered, but a slight one upon the unfiltered urine.

5th Sample:—Urine passed on the 10th of September; on the 17th inst. no scum on the filtered, but a very decided one upon the unfiltered.

6th Sample:—Urine voided on the 14th of September; on the 20th inst. a very decided scum upon the unfiltered, but none upon the filtered urine; on the 22d inst. still no scum upon the filtered portion.

It is thus evident that the removal of even a portion of the animal matter contained in urine, exercises a very marked influence over the development of the fungus, and there is no doubt that if it were more completely separated the results would be still more obvious.

The separation of the whole of the nitrogenised matters almost invariably present in urine, can seldom be effected by filtration; nor do I know of any unobjectionable means by which, without altering the chemical condition of the fluid, it may be removed; were it in any case completely abstracted, it is certain that no development of the fungus would take place.

The condition, then, of the development of the fungus is likewise, to some extent, an indication of the amount of animal matter, especially albumen, contained in the urine; if the fungus be in patches only, then it is certain that the quantity of nitrogenous matter is but small, but if the fungus form a continuous stratum over the whole surface, then it may be inferred that the amount is considerable, sufficient indeed to excite a *suspicion* of the presence of albumen.

The necessity for the presence of animal matter is shown by the fact, that when a little albumen is added to any slightly acidulated solution, the same fungus as that ordinarily met with in the urine makes its appearance in the course of a few hours; without such addition the solution might be kept for any length of time, and no development of the fungus would occur.

A second condition necessary to the growth of this fungus is therefore the presence of animal matter.

But there are still probably other conditions requisite. The abundant growth of this fungus in bottles nearly filled with urine and corked, would appear, at first sight, to show that atmospheric air was not necessary, and from this fact it certainly appears that a very free or large supply of air is not required.

That some portion of air is, however, indispensable, is shown by the following circumstances:—1st. If the bottle containing the urine be well corked, and filled within a

very short distance of the neck, the development of the fungus will be retarded, and sometimes altogether prevented. 2d. Occasionally it has happened to me to notice that, after the removal of the cork for a minute only and the admission of air, the plant, which had previously been in a stationary condition, has grown surprisingly. 3d. The fungus will not grow in an atmosphere of carbonic acid, an experiment which may be easily tried by means of a bell jar filled with urine, inverted, and into which a small quantity of carbonic acid has been passed.

A third condition, then, is the presence of a certain amount of atmospheric air, or, rather, of the oxygen of which the air is in part constituted.

We have next to inquire, is the fungus so frequently found in urine a new species, or is it identical with one already known and described? From a careful comparison of this plant, in the several stages of its growth, with the well known *Penicilium glaucum*, it becomes evident that the fungus common to the urine is that species. *Penicilium glaucum* is a very common fungus, and is that which imparts the mildewed appearance so frequently presented by a variety of decaying vegetable and animal substances.

It is now proper to mention, that some of the particulars above referred to in the account given of the conditions necessary to the development of penicilium glaucum, are not altogether new.

That an acid fluid and albumen were conditions essential to the growth of this fungus was first made known by Dutrochet,[1] who recognised the plant, however, only in its filamentous state. These conditions have subsequently been further elucidated by MM. Andral and Gavarret,[2] who also have given a much more complete account of the development of penicilium glaucum, than existed up to the

[1] Mémoire pour servir à l'Histoire Anatomique et Physiologique des Végéteaux et des Animaux, t. ii.

[2] Recherches sur le développement de Penicilium glaucum sous l'Influence de l'Acidification dans les liquides Albumineux Normaux et Pathologiques, 'Annales de Chimie,' t. lxxxiii, p. 385.

time of the publication of their memoir. These well-known observers were likewise the first to show that the presence of atmospheric air, or rather oxygen, was necessary to its growth; this they did by replacing the air over the surface of the fluid with carbonic acid; the development of the plant was entirely arrested for ten days, when the air being readmitted or oxygen supplied, the growth proceeded as before.

The fluids experimented upon by MM. Andral and Gavarret, were the serum of the blood, white of egg, the serosity from the péritonæum, from a hydrocele, and from blisters, also pus; the urine, the most important and interesting in a pathological point of view of all the animal fluids, being so entirely overlooked as not once to be alluded to even in the whole course of their investigations; lastly, one of the conditions laid down by these observers as essential to the development of penicilium glaucum, is really not so, since the fungus makes its appearance and grows in acid solutions containing animal matter which is not albuminous; as, for example, the aqueous humour of the eye diffused through water, a substance which is not coagulable by heat.

From a review, then, of the whole of the facts and observations above recorded, the following conclusions may be deduced:

1st.—That there is very frequently developed in urine a species of fungus known by the name of penicilium glaucum.

2d.—That this fungus ordinarily passes through three stages of development, any one of which is characteristic of the species; it exists first as sporules, these pass into thallus, and from this proceeds the perfect or aërial fructification.

3d.—That the conditions necessary for the development of this plant are, animal matter, especially but not exclusively albumen, an acid solution and oxygen, its growth being likewise much influenced by temperature.

4th.—That it may be developed at will in a variety of

other animal solutions besides the urine in which the above conditions are fulfilled, as in solution of white of egg, acidified with acetic, phosphoric, or any other acid.

5th.—That one of these conditions, viz. the presence of albumen, exists in almost all urines, whether neutral, alkaline, or acid.

6th.—That inasmuch as one of the requisite conditions is wanting in neutral and alkaline urines, the fungus never makes its appearance in these, no matter how much albumen they contain.

7th.—That the plant may, however, be developed at will, in even neutral and alkaline urines, simply by rendering such urines acid by means of phosphoric or any other acid.

8th.—That its presence may be regarded as, to some extent, an indication of the degree of acidity of the urine.

9th.—That it is not characteristic, as has been supposed, of the presence of an abnormal quantity of albumen in acid urines, since it is frequently developed in many urines which contain only a normal amount of epithelium and mucus, and in which also not a trace of albumen can be detected by means of heat and nitric acid.

10th.—That, nevertheless, it affords some indication of the amount of animal matter contained in acid urines; for where this is large, the fungus is usually developed in considerable quantity, and in all such cases it is proper that the urine should be tested for albumen.

11th.—That this fungus is no indication whatever of the presence of sugar in the urine, since the observations above recorded were all made upon non-saccharine urines, and since the fungus may be developed at will in solutions which it is certain do not contain a particle of sugar.

I have now to remark, in bringing the *first part* of this communication to a conclusion, that the observations detailed on the development of penicilium glaucum in the urine under different conditions, were made principally in the summer and autumn of the year 1849.

Part II.

I come in the next place to the consideration of the second division of the subject—viz. the development and growth of Torulæ in saccharine urine.

From the quotations given in the first part of this communication, it appears that, up to the present time, great difference of opinion prevails as to the value of the torula-test as an indication of sugar in the urine—some asserting that it affords positive evidence; others denying altogether that it is a reliable test. From the facts and observations already advanced, it is at least certain that torulæ in urine are not, in all cases, indicative of the presence of sugar. It has yet to be determined, however, whether the torulæ contained in saccharine urine are not characterised by such peculiarities as constitute a satisfactory test for sugar when present in that excretion.

For the determination of this point, a number of samples of diabetic urine were placed in distinct vessels, the changes which ensued being observed from day to day. Each sample was divided into two, and sometimes three, portions.

The first was placed in a glass, and freely exposed to the air; the second, in a bottle, air being admitted to a limited extent only through an aperture in the cork; and the third was enclosed in a tightly-corked bottle.

The following changes were observed by the eye alone to occur in that portion of the urine which was exposed to the air:—

In the course usually of two or three hours after being voided, the urine began to lose its transparency, and to present a milky appearance.

At the end of from 24 to 36 hours, cloudy, gelatinous-looking masses appeared suspended in the urine just beneath the surface, but extending some depth into the fluid. Although visible on the surface, the form and size of the masses were best seen from a side view. These masses, being exceedingly soft and delicate, quickly broke into

pieces on the least disturbance of the glass, and slowly subsided to the bottom; the same thing happened occasionally when the masses had attained considerable size—an inch or more in diameter—even when the urine remained undisturbed. Thus, after a few hours, the exact time varying according to temperature, there was an accumulation of these gelatinous-looking masses, not only on the surface of the urine, but also at the bottom of the glass, forming a cloudy sediment, the turbidity of the whole urine now having become very considerable.

Entangled in the masses, particularly those near the surface, were numerous bubbles of gas; these, separating from time to time, escaped into the air. Many bubbles were also thrown off from the masses at the bottom of the vessel, rising slowly to the surface; occasionally a number of globules became developed in these masses, which ascending, carried with them the masses in which they were included.

This elimination of gas was continued for some days, and was so great as clearly to indicate an active fermentation in the fluid. The gas generated was ascertained, by the following simple proceeding, to be carbonic acid gas:

Two or three ounces of the urine were placed in a wide-mouthed glass jar. In this a second vessel filled with lime-water was suspended, the mouth of the jar being well secured. The lime-water was soon observed to become turbid, and at the end of two days a considerable deposition of a white powder had taken place in the inner vessel. This precipitate effervesced on the addition of acetic acid, showing that the gas which had escaped from the urine and combined with the lime was the carbonic acid gas. A modification of this experiment was performed by means of a *Woolfe's* apparatus.

One of the flasks was partly filled with the urine; the other, with lime-water. A bent glass tube adapted connected the two, one end dipping into the lime-water. The gas, as it was evolved from the urine, passed through the tube, rendering the lime-water turbid, and producing a

precipitate, which effervesced as before on the addition of an acid.

For the next two or three days, reckoning from the end of the first 36 hours, the urine continued to present nearly the same character, except that the masses increased in size and number, became whiter, and acquired greater consistency; the globules of gas eliminated also becoming larger and more numerous.

At the end of about the fifth or sixth day the gelatinous masses had disappeared, some having subsided to the bottom, while others had gradually merged into and formed a continuous stratum of a fawn colour, having, to a certain extent, the consistence and characters of beer-yeast.

This stratum, from day to day, acquired increased firmness; so that, at the end, usually of seven or eight days,—sometimes earlier,—it might be removed as a distinct and coherent layer. By degrees its texture became altered, and it soon presented a woolly and filamentous appearance. Lastly, a crop of delicate transparent threads sprang up from the surface, bearing on their summits minute spherical heads of a black colour, barely visible with the naked eye.

In the course of a few days, the stratum, now of considerable thickness, gradually altered in colour—became brownish—and, after a further time, soft and brittle, ultimately breaking up and sinking to the bottom of the glass.

These changes, visible with the eye alone, are so marked and peculiar, that when once carefully noted, they cannot be mistaken. But there are still other more important changes and peculiarities corresponding with the several outward changes above described, and for the determination of which the microscope is necessary.

Examined with that instrument, the cloud-like masses were found to consist of the minute sporules of a fungus, imbedded in a mucoid base. These sporules were very irregular in size; some, when viewed with an object-glass of $\frac{1}{8}$th of an inch focus, being visible as mere black points, while the largest did not exceed the $\frac{1}{6750}$th of an inch in

diameter. These masses, composed of the minute sporules, constitute *the first sub-stage* of the development of the fungus. (Pl. II, figs. 1, 2.)

The soft, fawn-coloured scum is composed, for the most part, of circular sporules many times larger than the former. These, although usually separate, are occasionally feebly united in rows formed of two, three, or even more sporules; sometimes the sporules collect together in groups, the smaller surrounding the larger, or parent-cells. Intermixed with the sporules are also a few jointed and beaded threads.

These sporules, like the former, vary considerably in size, the smallest being scarcely the $\frac{1}{1900}$th of an inch, whilst the largest are as much as the $\frac{1}{628}$th of an inch in diameter, the medium size being the $\frac{1}{922}$d of an inch. (Pl. II, figs. 3, 4. Pl. III, fig. 1.)

Between these sporules and those first described, it will be observed a considerable difference of size exists; this, for a time I was at a loss to explain; the explanation is furnished, however, by a consideration of the manner in which the sporules are developed.

The sporules are multiplied by the constant escape, from the interior of the larger sporules, of other and smaller cells, these, on their escape, appear on the surface as buds, and are usually included in a pouch-like protrusion of the parent-cell wall, which with its contained nucleus, is finally thrown off, becoming a new and independent sporule.

This evolution of sporules at the early period of the development of the fungus, is so rapid and continuous as not to allow any of the sporules to attain a large size. Subsequently, however, as the quantity of sugar becomes diminished, the evolution is less rapid, and time is afforded for a large proportion of the sporules to acquire the size characteristic of the fungus in *the second sub-stage* of its development.

The more consistent stratum is made up of branched and jointed threads, intermixed with a few separate circular sporules. These threads are frequently beaded, the beaded

cells being sometimes placed in the course, but more frequently forming the termination of the threads. In the latter case, the beaded extremities are often raised above the surface of the urine, and project a short way into the atmosphere. (Pl. v, fig. 1. Pl. III, fig. 2.)

Not unfrequently single cells several times larger than the others are observed; these are placed in the course of the beaded portion of the threads; but sometimes they are seen lying loose; these cells appear to be of the nature of vesicles. (Pl. III, fig. 3.) This forms the second stage of the development of the fungus, that of thallus.

The stratum presenting a woolly structure is divisible into two parts; the one, rests upon and is immersed in the urine; the other, projecting into the air, may be termed aërial. The first consists principally of the thallus above described, while the second is made up of the slender, transparent, jointless, and occasionally branched stems which here bear the globular heads.

The state and appearance of the heads vary with the development. At first they present a smooth outline from being covered with a delicate membrane. (Pl. v, fig. 2.) This afterwards bursting and becoming retracted, a rounded mass of circular sporules of a brownish colour is disclosed to view. The sporules falling off, leave the dilated extremities of the threads or filaments exposed.

These changes constitute the third or perfect stage of development of the fungus, that of aërial fructification.

The rapidity with which the fungus is developed is dependent, to a great extent, on temperature; heat, as the warmth of summer, greatly accelerates, while cold retards the growth to an equal degree. So much is this the case, that it is doubtful whether the sugar fungus would be developed at all in mid-winter, and when the thermometer was below the freezing point.

The observations upon the development of the diabetic fungus, above recorded, were made during the summer months; the periods given are those which were found to correspond to the several stages of the growth of the fungus at

that season of the year. It must be remembered, however, that the development is influenced considerably by variations of temperature, even in summer.

Although the appearances above described were all noticed in the first sample of saccharine urine subjected to observation, yet a variety of other samples, which were afterwards submitted to similar investigation, furnished results in all essential respects identical.

Such is a brief description of the changes which ensued in samples of saccharine urine exposed to the atmosphere. We have, in the next place, to notice those changes which occurred in the two other portions of the first urine, to one of which air was admitted to a limited extent, and from the other entirely excluded.

The portion of urine partially excluded from contact with the air quickly became, like the first, whitish and opaque; the cloud-like masses appeared as before, broke up on the slightest motion, and subsiding, formed a copious sediment. Many globules of carbonic acid gas arose from all parts of the liquid, and after accumulating on the surface escaped into the atmosphere. The masses were, however, fewer and smaller than in the sample freely exposed to the air, and the globules of gas were much less numerous, and their evolution ceased at an earlier period.

Examined with the microscope, the masses were ascertained to consist, as in the first portion, of myriads of minute sporules.

With the formation of the masses, the development of the fungus ceased; the only ulterior changes being, that the masses gradually became whiter, and more consistent.

The sporules, no matter how long the urine was kept, never attained the large size which distinguishes them in a more advanced condition of development.

The urine contained in the closed vessel was turbid when introduced; this turbidity afterwards increased somewhat, and bubbles of carbonic acid gas became evolved here and there. At the end of a few hours, however, the weather being extremely warm, the bottle burst with a loud ex-

..., breaking into many pieces, which were scattered ... and near; the liberated urine effervescing on its escape, as though it were so much ginger beer. The same occurred in a second sample; but in other trials, this result was obviated by employing a Woolfe's apparatus. One of the flasks was partly filled with lime water, which, by absorbing the gas as quickly as generated, removed the pressure, and so prevented the bursting.

The changes which took place could now be readily noted: the urine, slightly turbid at first, soon became more opaque, and some carbonic acid gas was evolved, yet its opacity was soon lost; the elimination of gas ceased, and ultimately it became perfectly transparent. The few minute sporules which were originally diffused throughout the liquid fell to the bottom, forming a slight sediment, and for whatever period the urine was kept, no gelatinous masses were developed in it, nor was any stratum of fungus formed.

From the great differences observed in the fungus in the several portions of urine, it is very evident that free exposure to the air is a condition indispensable to its perfect development; deprived of this, its growth is quickly arrested.

It is also very evident from the description and illustrations now given, that the fungus developed in saccharine urine is a species very different from that treated of, in the first part of this communication, viz., *Penicilium glaucum*.

Further, a comparison of the diabetic fungus with the yeast plant, shows that the two are identical; a point of very considerable interest. The figures which accompany this communication contrasted with those of the yeast plant, published in the 'Lancet,' vol. i, 1850, are in themselves sufficient to establish this identity.

Up to a very recent period, great uncertainty, and even mystery, hung over the development of the yeast plant; the efforts made by able observers, to trace it through all the phases of its development, having for the most part completely failed.

In the communication referred to,[1] I gave a description of the yeast plant; and traced it through several stages of development; I followed the transformation of the sporules into branched threads, or thallus; detected the beaded threads and the large sporangia-like cells; and at that time thought I had really traced it, step by step, to its final condition. I have since ascertained, however, that under favorable circumstances, perfect aërial fructification is produced, precisely similar to that described as constituting the last and perfect stage in the growth of the diabetic fungus.

Now the changes described, as occurring in the three portions of the same sample of diabetic urine placed under such opposite circumstances, were with slight differences repeated in a variety of other samples, some obtained from patients labouring under diabetes in different degrees. So there is no doubt, but that these changes, under similar conditions, are constant, and therefore they afford valuable and unmistakeable evidences of the presence of sugar in the urine.

It is not to be understood, that the whole of the changes described as occurring in diabetic urine, were fully appreciated from the observation of a single specimen, and that the first submitted to examination. It was necessary in order to arrive at all the results above recorded, to watch the changes which ensued in a variety of samples; but these changes having once been clearly ascertained, the whole of them were readily afterwards followed out in even single specimens.

I will now proceed to give the results, recorded from day to day, derived from the observation, as well as chemical and microscopical examination, of several samples of diabetic urine, in order that the precise and positive character of the facts upon which the description contained in the foregoing pages is founded, may be the more clearly comprehended.

[1] Bread and its Adulterations, 'Lancet,' April, 1850.

Results recorded from day to day, of the Examination of Samples of Diabetic Urine.

1st SAMPLE:—This urine was passed on the morning of the *7th* of *June*, 1852, but did not come into my possession until the 11th inst., it having been kept in a corked phial; it was very acid, had a specific gravity of 1037, and examined with the microscope there were detected in it numerous octohedra of oxalate of lime; it was divided into two portions.

1*st portion in open vessel.*—Examined on the 11th inst. There were observed near the surface of the urine a few cloud-like gelatinous masses composed of myriads of minute sporules imbedded in a mucoid base. Sporules of *Penicilium glaucum*, some round, but the majority of an oval form, were likewise noticed resting on the urine.

Examined on the 13th inst. There were seen on the surface with the naked eye a few small circular patches of Penicilium glaucum, composed of sporules, some round, but the greater number oval, while at the bottom of the glass was an abundance of sporules, both small and large, of the saccharine torulæ, as well as a few filaments of the same, some with bearded cells.

Examined on the 19th inst. The Penicilium was still in the same state, but a thick white woolly stratum of the diabetic fungus had become developed, forming a ring round the whole margin of the glass; this consisted principally of the thallus; that is, of the root-like portion of the plant, which is made up of branched and bearded threads; intermixed with the filaments were, however, numerous large sporules, and from the upper surface of the stratum a considerable number of straight filaments shot up.

Examined *June* 23*d*. The woolly stratum now extends nearly over the whole surface of the urine; and the vertical threads are seen by the eye alone to bear on their summits the minute spherical and black heads which are cha-

racteristic of the fungus in its perfect state. (See Pl. v, fig. 2.)

Examined *July* 3*d*. The globular heads have lost the smooth outline which they at first presented, and they now consist of masses of sporules of a rounded form and deep brown colour, supported on the extremities of the vertical filaments; in some cases, the sporules have fallen off, the dilated extremities of the filaments then coming into view. The stratum breaks up readily; and on replacing it in the glass from which it had been removed for a few minutes, it sank to the bottom.

Examined *July* 19*th*. Stratum risen again, and spread over the whole surface of the glass; patches of Penicilium glaucum in perfect fructification have appeared; the diabetic torula now seen is chiefly the results of a second development, which, like the first, has passed through all the stages, even the last, that of aërial fructification.

The urine is now pale, but thick and turbid, as though mixed with flour; and there is a copious deposit, consisting principally of the sporules of the diabetic fungus: it is alkaline, contains an abundance of triple phosphate, and the potash and copper tests furnish no results, showing that the sugar has at length disappeared.

2*d portion in closed vessel.*—Although passed on the 7*th* of *June*, this portion was not placed in the closed vessel until the 11th inst., the saccharine torula had therefore become developed to some extent previous to the exclusion of the atmosphere.

Examined on the 13*th* of *June*. Urine in the same condition, and containing the same structures as were detected in the portion exposed to the air at the same period, the only difference being that there was very much less of the saccharine torula.

Examined 19*th June*. No increase in the quantity of saccharine torula, and none present at the top of the urine, the pellicle of Penicilium on surface in the state of oval sporules.

Examined 3*d July*. Saccharine torula in the same state, no scum of Penicilium on surface.

Examined 18*th July*. Urine pale, perfectly clear, and possessing a strong acid reaction; still contains traces of sugar.

Examined 16*th September*. Urine bright, clear, and still very acid; the sugar has now disappeared entirely.

2d SAMPLE:—*Placed in partially closed vessel.* Passed 22*d May*, but did not come into my possession until some time afterwards, it having been kept in a corked phial. When examined with the microscope, there were detected in it at the bottom a few sporules, both large and small, of the saccharine fungus, hexagonal crystals of uric acid, and octohedra of oxalate of lime, (see Pl. III, fig. 3.) It was placed in a partially closed vessel on the 14*th June*.

Examined 19*th June*. There was an abundant gelatinous frothy scum on surface, consisting of the small sporules of the diabetic torula, and of numerous bubbles of carbonic acid: there was also a considerable deposit of the small sporules, intermixed with a few of the large ones at the bottom of the vessel.

Examined 3*d July*. Gelatinous and frothy scum nearly all subsided to the bottom, the sediment consisting, as before, of the minute sporules, with a few large ones; urine still acid.

Examined 18*th July*. The thick frothy gelatinous scum had reappeared on the surface, but on shaking the urine it again fell; there is now a very considerable deposit divisible into two layers, the lower of a fawn colour, consisting of the small and large sporules of the saccharine torula, and the upper of the small sporules only: the smell of the urine is sour and acetous, but the reaction slightly alkaline; sugar gone entirely.

Examined 8*th August*. The crystals of uric acid have disappeared, and their place is supplied by numerous globules of some urate: small sporules of the diabetic fungus may still be detected. It will be observed that neither in this nor the previous specimen did the saccharine torula attain its full development.

3d SAMPLE:—*In open vessel.* This urine did not reach

me until some time after it had been passed; it was when received, however, of highly specific gravity, acid, and contained a considerable quantity of sugar. Exposed to the air for some days, the surface became covered with the thick woolly stratum, which, on examination, was found to consist of the diabetic torula in its perfect condition.

4th SAMPLE:—*1st portion in open vessel.* Urine passed *23d July*, 1852; specific gravity 1033, acid.

Examined *July 24th.* Copious gelatinous-looking flocculi, with many bubbles of carbonic acid imbedded in and surrounding them, have appeared near the surface of the urine; these consist of vast numbers of minute circular and oval sporules, immersed in a mucus-like base. The urine has a milky or floury appearance, which is occasioned by the great numbers of sporules diffused throughout.

Examined *25th July.* Flocculi increased in size, and many have fallen to the bottom, bubbles of carbonic acid gas are seen rising from all parts of the urine to the surface.

Examined on the *27th July.* Nearly in the same state.

Examined *30th July.* Surface covered, particularly at the edges, with a thin plicated scum of Penicilium, which consists of oval sporules, some extending into short threads. No large sporules of the saccharine torula have as yet appeared; the urine is still very acid, and has a specific gravity of 1024.

Examined *8th August.* The plicated thin scum, consisting of the sporules and threads of Penicilium, is still seen at the sides; and in the centre a large mass raised above the surface, and also extending much beneath it, having the consistence and colour of yeast: this mass consists principally of the large sporules of the saccharine torula, and which are not distinguishable under the microscope from those which form the yeast plant; the urine is thick, as though flour were diffused through it, very acid, and still contains sugar, but a small quantity, judging from the action of the potash test.

Examined *8th August.* The perfect fructification of the

Penicilium has now become developed, forming a green circle round the yeast mass. The further changes which ensued in this sample were not followed.

2d portion in partially closed vessel.—Examined on the *24th July*. This urine is in the same condition as the specimen in the open vessel at the same date.

Examined *25th July*. Same state as the previous specimen.

Examined *8th August*. A thin pellicle of Penicilium on surface, consisting of sporules intermixed with a few short threads; gelatinous masses both on the surface and at the bottom of the vessel, a very few diabetic sporules of large size being detected in the latter situation: specific gravity 1022, very acid, and contains more sugar than the urine exposed to the air.

Examined *17th September*. The masses near the surface have become whiter and more consistent, and there is a very considerable deposit of the same. With the microscope the masses were found to consist of the sporules of the saccharine fungus, both large and small, but chiefly the latter, mixed with a few broken filaments: resting on the flocculi near the surface were many fine crystals of oxalic acid. This urine has a smell like that of sour milk, is very acid, and still contains a little sugar.

3d portion placed in closed vessel.—Urine a little thick and white, as though mixed with flour; bubbles of gas rising from all parts to the surface, showing that it is on the work: bottle burst the same day with a loud explosion, the fragments being scattered here and there, and the urine effervescing like so much ginger beer.

5th Sample:—*1st portion placed in open vessel.* Passed *2d August:* the urine became milky almost as soon as voided, from suspended sporules of the saccharine torula; it also quickly threw up a large quantity of carbonic acid gas.

Examined *8th August*. Scum of torula on surface composed, in part, of the oval sporules and branched threads or thallus of Penicilium glaucum, and in part of the small sporules of the saccharine fungus: at the bottom of the

vessel there was a considerable deposit formed by the small diabetic sporules only. At this date the urine was very acid, and still contained sugar, although it only had a specific gravity of 1006: the density of the urine when first passed was not ascertained; it was most probably of low specific gravity, however.

Examined 14*th August.* Diabetic torula in nearly the same condition; sugar all disappeared. The fungus in this instance did not pass through all the stages of its development, in consequence of the early and rapid transformation and disappearance of the sugar.

2d portion in partially closed vessel.—Examined *August 8th.* Very acid, specific gravity 1004: thin scum of torula on surface formed of the sporules of the saccharine fungus and of Penicilium glaucum intermixed; a considerable deposit of the same.

Examined 17*th August.* Saccharine fungus in the same state; urine smells very sour, and is strongly acid.

Examined 17*th September.* A thin brownish scum of torula on surface, composed chiefly of the small sporules of the saccharine fungus: urine gelatinous-looking, very acid, and of a sour smell; does not now contain sugar.

3d portion in closed vessel.—The urine was placed in the closed vessel the day on which it was voided; the next morning it was milky, and many bubbles of gas had collected on the surface: in the course of the day the vessel burst with a loud noise, the urine effervescing briskly from the large quantity of carbonic acid gas set free.

6th SAMPLE:—1*st portion placed in open vessel.* Urine passed 7*th August,* of specific gravity 1028, became somewhat milky shortly after being voided.

Examined 8*th August.* Several gelatinous-looking masses had formed on the surface, where also large numbers of bubbles of carbonic acid gas had collected; the subsequent changes were the same as in the other samples freely exposed to the air; the fungus continued to grow until it reached its complete development.

The changes which occurred in the other two portions of

the same urine, the one partially exposed to the air, the other excluded from it, were so nearly similar, that it is unnecessary to describe them. Sufficient details have now been given to show the precise character of the alterations which ensue in specimens of saccharine urine placed under different conditions.

But it may be said that there are already tests sufficient of the presence of sugar in the urine; and, therefore, although the torula-test is very satisfactory, yet that it is not needed. To this objection I next reply—

There is no doubt but that in cases of confirmed diabetes, where the quantity of sugar in the urine is very considerable, the potash and copper tests afford positive indications; but do they in slight and incipient cases of that disorder?

The physician is not unfrequently consulted in supposed cases of diabetes, the symptoms being—loss of health, emaciation, but particularly an elimination of an increased quantity of urine; and yet, failing when he comes to test the urines by the ordinary reagents to discover the presence of sugar, he generally pronounces these cases not to be diabetic. Does he, in this way, always arrive at a correct conclusion?

The detection of diabetes in an early stage, where sugar is present in the urine, either occasionally only, or in small quantities, is of the highest importance; for it is then chiefly that the physician may entertain the hope of treating the disorder successfully.

In an article published in the 'Lancet,'[1] I showed that diabetic sugar might be introduced in quantities by no means inconsiderable into many different urines, and yet not be discovered afterwards by the most skilful application of the ordinary tests.

Now, this fact confirms in a remarkable manner the suspicion entertained by many that urines may contain small quantities of sugar, and yet that this shall not be detected by any of the methods ordinarily in use.

[1] On the Tests for Sugar in the Urine, 'Lancet,' 1851.

I have now ascertained that this is not unfrequently the case.

Several specimens of urine voided in a supposed case of incipient diabetes were set aside for observation, they having previously been carefully tested for sugar, but none having been discovered.

In some of these specimens, somewhat to my surprise, although such a result was not of course wholly unanticipated, the gelatinous masses previously described appeared, bubbles of carbonic acid were eliminated, and the diabetic torula or fungus was traced through all the stages of its development—even the last, that of perfect aërial fructification.

The only differences observed in the development of the fungus in these specimens contrasted with its growth in samples of urine containing large quantities of sugar, were in the size and number of the masses, which were fewer and smaller, in the thinness of the yeast-like stratum formed, and in the circumstance that this, as well as the perfect fructification which sprang from it, did not cover the whole surface of the liquid, but extended over part only, forming one or more patches.

In other specimens development entirely ceased at the end of the first stage, the urine became turbid, the gelatinous masses were formed, and carbonic acid evolved; but here the growth stopped—the masses broke up, and after a time disappeared.

Lastly, in other specimens, the diabetic torula did not make its appearance at all.

It was particularly noticed that those specimens in which the fungus went through all the stages of development were more than usually acid.

That those urines in which the development ceased quickly were but feebly acid when passed, the acidity soon being entirely lost.

Finally, that the urines in which the fungus did not make its appearance at all were frequently either alkaline when voided or very quickly became so.

It appears, then, that in the diabetic fungus we have a most valuable, and, indeed, the only certain and available, test of the presence of sugar in urine in small, but not inconsiderable quantities.

It has been remarked that it was only in the more acid samples that the fungus became fully developed. This may be readily accounted for.

When describing Penicilium glaucum, I stated that the conditions necessary for its development were free exposure to the atmosphere, albumen to act as a ferment, and an acid liquid. Now, the same conditions are requisite for the growth of the diabetic fungus, with the addition of a fourth —the presence of grape or diabetic sugar.

In the feebly acid or alkaline urines one of these conditions is not fulfilled, and, therefore, the fungus is not developed.

It may be said, however, that urines which contain sugar are always acid, and therefore, that the fungus should be developed in all cases where this is present. Where the quantity of sugar is very considerable the urine no doubt is constantly acid; but whether it is always so, where the amount is small, is less certain. With a view to determine this point, I adopted the following proceeding:

Several samples of the feebly acid or alkaline urine passed by the patient the subject of incipient diabetes were obtained; to these was added sufficient phosphoric or acetic acid to impart the decided acidity necessary for the development of the fungus should sugar be present. The specimens were watched from day to day, and as any lost their acidity, as sometimes happened, further quantities of acid were added. This proceeding furnished the following results:—In the whole of the samples the circular patches of Penicilium glaucum quickly made their appearance, ultimately passing through all the stages of their development. In one of the samples only was there any formation of the sugar fungus, and in this the growth did not proceed beyond the stage of large circular sporules. One would, therefore, be disposed to conclude as the result of this

experiment that sugar is not ordinarily contained in slightly acid, neutral, or alkaline urines.

In suspected cases of diabetes, then, should the fungus not appear in the first specimens of urine examined, it must not be concluded that sugar is not present, even although the urines possess some degree of acidity. We must ascertain whether they are sufficiently acid, and, if necessary, must increase that acidity; neither must we decide against the presence of sugar in those instances in which some of the samples of urine examined are alkaline, for, as is also shown above, sugar may be present in some and absent in other specimens. In the case of incipient diabetes, which I have made the subject of special observation, I have particularly noticed that sugar is most liable to occur in the urine voided after error and excess in diet.

In this place the observations of Dr. Basham, 'On the Cholera Sporules,'[1] may be referred to. While searching the urine of a dyspeptic patient for crystals of oxalate of lime "the appearance of some annular-formed cells attracted attention, some with minute nuclei. The field of the microscope presented these sporules amongst many crystals of the oxalate and some epithelium and mucous globules." Again, in examining, in another case, some urine which was strongly acid, abounded in stellated crystals of uric acid, and was of specific gravity 1018, Dr. Basham observed some sporules which he thus describes: "They are somewhat like the torula of diabetic urine; but they want the true confervoid character. They are oval cells, arranged by their long diameter in a bead-like form, with minute granules or cellules developing themselves from the surface and junction of the parent-cells."

Dr. Basham made pen-and-ink sketches of the appearances observed at the time. These are published in the third edition of Dr. Golding Bird's work, and from an examination of fig. 58, which represents the character of the fungus detected in the second sample of urine examined, I entertain no doubt whatever but that this drawing

[1] Medical Gazette, 1849, vol. xliv, p. 686.

exhibits a condition of the sugar fungus. The urine, therefore, although not of high specific gravity, contained a small quantity of sugar, to which the presence of the torula was doubtless due. This case affords further evidence of the value of the torula-test for sugar in the urine.

From the facts which I am now about to adduce, it becomes extremely probable that sugar frequently makes its appearance in the urine in connection with a more or less alkaline condition of that fluid. It is at all events certain that it sometimes does so.

For some years before, and up to the period of the discovery of sugar, the urine in the case of diabetes, so often referred to, was when first passed occasionally alkaline or neutral, but most frequently feebly acid. On becoming cold, and even while still acid, it usually threw up an abundant iridescent pellicle of phosphate of magnesia; and when cold, it deposited large quantities of triple phosphate. One of the consequences of this want of acidity was that Penicilium glaucum, one of the best tests of acidity, but seldom became developed in it, and, when it did appear, it still more rarely passed through all the stages of its growth.

But the most remarkable character of this urine was, and still is, that it frequently contains very large quantities of phosphate of lime in a crystallized state. Now, this earthy salt occurs but very rarely in the urine in this condition, and of it, so far as I am aware, no accurate or detailed description has yet been given. I have myself met with crystals of phosphate of lime in several different cases; and in 1850 I published in the 'Lancet' a short notice of the form and composition of certain modifications of the crystals of this phosphate.[1]

Although this salt has not yet been fully described, we yet find in works on organic chemistry one or two brief references relating to it.

Thus, in vol. ii, p. 133, of Simon's 'Animal Chemistry,'[2]

[1] On certain Important Points in the Chemistry and Pathology of the Urine, 'Lancet,' January 19, 1850.

[2] Translation by Dr. Day.

the following observation occurs: "The phosphate of lime may be recognised under the microscope as an amorphous mass. Sometimes, but rarely, it occurs in a crystalline form. Both varieties are figured in fig. 26." In this figure a granular powder, as well as certain foliaceous masses, are represented. The form of these is so irregular that it is impossible to refer them, with anything like certainty, to the crystals I am about to describe; while, appended to the explanation of the figure, a remark is added to this effect — the foliaceous bodies are most probably urates. It thus appears that Simon was himself in some doubt respecting the composition of the irregular bodies which fell under his observation.

Again, Dr. Griffith, in his little work, copies Simon's figure, and adds, "I have specimens of this."[1]

Lastly, I find crystals of phosphate of lime described and figured under the name of "*Penniform Crystals of the Neutral Salt*," by Dr. Golding Bird. The description is as follows: "This very elegant variety of the neutral magnesian phosphate has only lately fallen under my notice, and has occurred in a very few cases. It presents the appearance of striated feather-like crystals, two being generally connected, so as to cause them to resemble a pair of wings. (Fig. 35.) I cannot give any satisfactory explanation of the causes of this curious and elegant variety, or whether they differ in any way chemically from the prismatic form. The few specimens I have met with occurred in acid urine."[2]

I found my opinion that the crystals thus described by Dr. Bird are not composed of the neutral triple phosphate, but of phosphate of lime, upon an examination of one of Dr. Bird's original preparations, kindly lent me, along with others, for the purpose of having figures made from them.

Since the occurrence of these crystals is of much importance, in more respects than one, but particularly in relation to the presence of sugar in the urine, I will describe the forms which they assume, and especially the

[1] Practical Manual. [2] Urinary Deposits, 2d ed., p. 212.

method of analysis adopted, so that no room may be left for supposing that a correct conclusion with respect to the composition of these crystals has not been arrived at.

First, I would observe the crystals have presented themselves to my notice in the urine for at least the last three or four years. Although generally present, especially when the health is more than usually affected, yet they are sometimes absent entirely,—it may be for days together,—or they may be absent from one specimen and present in the next. They vary also in number: sometimes there are but few; usually they occur in great abundance, particularly in the more acid samples, in which they are formed even while the urine retains a decided acid reaction, and long before the formation and deposition of the crystals of triple phosphate.

Viewed with a half or quarter inch object-glass, the crystals appear wedge-shaped—being broad at one extremity and narrow at the other; but when the $\frac{1}{8}$th inch glass is brought to bear upon the broad end of the crystal, which is the only completely-formed part, it is then seen that they are really six-sided prisms, with oblique, and sometimes dihedral, summits. Occasionally, but rarely, both ends of the crystal are perfect, and then the wedge-shaped appearance is lost, and both extremities are alike. Sometimes they occur singly, but the greater number usually form, by the union of several crystals by their narrow extremities, rosettes more or less perfect; in other cases, but this is very seldom, the crystals are compound, each breaking into numerous secondary crystals; when this occurs, both ends are generally of the same shape.

The crystals are formed first, and chiefly on the surface of the urine, but they are sometimes found in large quantities at the bottom of the glass, and even adhering to the sides; those on the surface are frequently imbedded in a crust of iridescent phosphate of lime.

The crystals were twice carefully analysed, being obtained for the purpose, in the following manner: after having been identified by means of the microscope, they were

skimmed off the surface of the urine, and repeatedly washed in distilled water, to free them, as far as possible, from impurity; it is rarely, however, that they can be procured in any quantity, entirely free from admixture with small quantities of either phosphate of magnesia, triple phosphate, or even both these.

In the first sample analysed, there were present a few crystals of triple phosphate, and a little phosphate of magnesia; the deposit thus contaminated exhibited the following characters: it was slowly dissolved by cold acetic acid, and very rapidly by hot; from this solution oxalic acid threw down a copious precipitate of oxalate of lime, when boiled with liquor potassæ ammonia was evolved; it was fusible with difficulty only before the blowpipe.

From the above reactions, it is evident that the crystals are composed principally, if not entirely, of phosphate of lime; the small quantities of magnesia and ammonia detected being derived from the triple phosphate and phosphate of magnesia, which were previously ascertained to be present; it is nevertheless possible, that the lime may be combined with a small quantity of ammonia.

The second sample was almost, if not entirely, free from the ammonio-magnesian phosphate, but it was admixed to a very small extent with phosphate of magnesia, animal matter in the form of vibriones, and perhaps with oxalate of lime.

On boiling a few of the crystals in a test tube with a little liquor potassæ, a small quantity of ammonia was evolved, which communicated a red stain, not permanent, to turmeric paper. After boiling for a quarter of an hour, the liquid was diluted and set aside; in a few hours, the clear supernatant liquid was poured off, then acidulated with acetic acid, and tested with lime for oxalic acid, on standing for two or three hours a faint precipitate of oxalate of lime formed. A little more of the deposit was then boiled with acetic acid, and the clear liquor tested for lime with oxalic acid, when an abundant precipitate was produced. After the precipitation of all the lime, the solution was super-

saturated with ammonia, and allowed to stand, when crystals of bibasic phosphate of magnesia and ammonia separated. Finally, heated before the blowpipe, the crystals melted with difficulty.

As the result of this analysis, it again appears that the crystals consist, for the most part, and in all probability entirely, of phosphate of lime. A trace of ammonia only was detected on this occasion, but very perceptible quantities of phosphate of magnesia and oxalate of lime, the former of these, and most probably the latter also, were present as impurities.

The question next arises, is there any connection between the crystals of phosphate of lime and sugar in the urine?

I have described these crystals as occurring most frequently and abundantly in the more acid samples of urine; now it is in precisely these that sugar most commonly makes its appearance.

Again, between sugar and lime there is a great and well-known affinity.

Lastly, lime is apt to occur in saccharine urine in another form, in combination with oxalic acid.

These considerations render it probable that there is some such connection; before, however, we shall be in a position to come to any definite conclusion on this point, further observations are required.

I will now give the results, as recorded from day to day, derived from the examination of specimens of the urine passed in the case of incipient diabetes.

Results of the Examination of Specimens of the Urine voided by the Patient the subject of Incipient Diabetes.

1st Sample:—Urine passed 5th August, slightly acid, having a specific gravity of 1015, clear when voided, and of a pale brandy colour, but becoming cloudy and thick as it cooled; flocculi separated from it, which subsiding formed a deposit three fourths of an inch in depth in a twelve-ounce

bottle; while the urine was being passed a slight smarting sensation was felt.

Examined *6th August.* The urine has now become alkaline, the deposit white and granular.

Examined *8th Aug.* A scum or pellicle has now formed over the whole surface of the urine, this consists of vibriones, a very few crystals of the ammonio-magnesian phosphate, and an immense number of crystals of phosphate of lime, mostly in stellæ, but some also single. The first analysis of the crystals given at p. 65 was made from this sample.

As at the time no suspicion was entertained that the case was one of diabetes, no search was made for the sugar fungus.

2d SAMPLE:—Passed early in the morning, on the *5th* of *August.* Urine clear, very acid; specific gravity 1024, and of a very deep colour.

Examined *8th August.* Many small circular patches of a variety of Penicilium glaucum in all stages, some composed of sporules, others of thallus, and some fully developed, and of a dark olive-green or brown colour; interspersed amongst these were a few white woolly tufts of Penicilium of larger size.

Examined *27th Aug.* Tufts of Penicilium in much the same state, but faded somewhat, and now imbedded in a pellicle of phosphate of magnesia. While examining one of the tufts under the microscope, many large sporules, as well as some of the threads forming the thallus of the saccharine fungus, were somewhat unexpectedly discovered, and it was afterwards ascertained that a considerable quantity of this fungus in an advanced state of development was present, not only on the surface, forming the tufts in part, but also at the bottom of the vessel.

Examined *5th September.* Urine thick and turbid, alkaline, with much deposit at bottom of glass.

Examined *17th Sept.* Urine dark brown; on examination of the tufts with the microscope, numerous blue masses were seen, but there were no crystals of phosphate of lime; the deposits consisted of vibriones, a great many spherules of some urate, and a few crystals of triple phosphate.

The saccharine fungus in this specimen did not reach its complete development.

3d SAMPLE:—Examined *5th September*. Passed about ten days since, but no record kept of its characters at that time. A thick yellow scum has spread all over the surface, with here and there imbedded in it a patch of fawn-coloured penicilium in perfect fructification, one patch somewhat green in the centre; this scum is composed of vibriones, phosphate of magnesia, and an immense number of the crystals of phosphate of lime, some separate, others in stellæ; the urine is still slightly acid, thick, and of specific gravity 1015.

Examined *17th Sept.* Urine alkaline; the scum first formed was removed, and the crystals of phosphate of lime after being well washed were submitted to analysis; a second scum similar in appearance has now collected on the surface, this is composed entirely of phosphate of magnesia, much triple phosphate, and vibriones, very small and active. At the bottom, there was present much phosphate of lime, a very small quantity of triple phosphate, and many vibriones.

There were also detected a few sporules of fungus, most probably those of the sugar torula.

4th SAMPLE:—This sample was also passed about ten or eleven days since; it is somewhat acid, and the surface is covered with circular tufts, in perfect fructification, of an olive-green fungus, a variety of Penicilium glaucum. The tufts are small and interspersed with several white woolly tufts of larger size, and not in fructification; spreading over nearly one half the urine is another fungus in perfect fructification, the saccharine; this is recognised by the long filaments which spring up on all sides, bearing on their summits the minute spherical heads which are so characteristic of the species. On examination with the microscope, numerous large sporules and threads of the diabetic fungus, as also many stellæ of phosphate of lime, were discovered,

intermixed with the threads forming the thallus of the olive-green tufts of Penicilium glaucum; the thallus of the woolly tufts likewise contained an immense number of stellæ of phosphate of lime.

5th SAMPLE:—Urine passed after dinner, on the *5th* of *September*, clear, of the colour of pale brandy, decidedly acid; specific gravity 1019; colour somewhat deepened by boiling with potash; no result with the copper test.

Examined *7th September*. Still clear and acid, has deposited much mucus, and some oxalate of lime.

Examined *8th Sept.* Turbid, but still acid, a few mucus-like masses near surface.

Examined *11th Sept.* Still acid; a decided scum on surface, composed of the small sporules of the saccharine fungus, vibriones, and a few crystals of phosphate of lime.

Examined *17th Sept.* Alkaline; saccharine fungus fallen to bottom, and still in the state of minute sporules; scum on surface, composed of much phosphate of lime and triple phosphate, with many vibriones. At the bottom of the glass, groups of sporules imbedded in masses of vibriones were detected, also very much oxalate of lime, phosphate of lime, and triple phosphate; the same also adhering to the sides of the glass in large quantities.

6th SAMPLE:—Urine passed early in the morning of the *6th* of *September*, clear, of a pale brandy colour, acid; specific gravity 1022; colour more deepened than in Sample 5, by boiling with potash; the copper test does not afford any evidence of the presence of sugar.

Examined *7th September*. Very turbid, decidedly acid, contains great numbers of octohædra of oxalate of lime, and much vesical epithelium.

Examined *8th Sept.* Urine very thick; cloud-like masses of the sporules of the sugar fungus both on the surface and at the bottom, as also many bubbles of carbonic acid gas.

Examined *17th Sept.* Urine alkaline; colour of urine

not deepened by keeping; scum on the surface, composed of crystals of phosphate of lime, a pellicle of phosphate of magnesia, and many vibriones; at bottom of glass, very much phosphate of lime, numerous globules of some urate, a little triple phosphate, vibriones, and a few dark sporules of fungus, perhaps, those of the sugar plant.

7th SAMPLE:—Urine passed after dinner, on the 6*th* of *September*, decidedly acid; specific gravity 1015; clear, and of a pale colour; colour deepened by boiling with potash; no evidence of the presence of sugar afforded by the copper test.

Examined 8*th September*. Very turbid, contains much vesical epithelium, but no oxalate of lime.

Examined 10*th Sept*. Feebly acid, smell a little offensive, very thick, with large masses of sporules near the surface, a considerable deposit of the same, and many globules of carbonic gas.

Examined 11*th Sept*. Scum on surface composed of vibriones, and phosphate of lime; no triple phosphate; urine nearly neutral.

Examined 21*st Sept*. No scum; crystals of phosphate of lime floating on surface, and adhering to sides, some compound, with both extremities perfect; triple phosphate, and a very few minute octohædra of oxalate of lime. At bottom, phosphate of lime, triple phosphate, and vibriones. No diabetic sporules of any size.

8th SAMPLE:—Passed after dinner, on the 6*th September*, slightly acid; specific gravity 1019.

Examined 8*th September*. Neutral, rather turbid.

Examined 10*th Sept*. Alkaline, still more turbid; deposit of mucus with much triple phosphate; on surface, a scum consisting of vibriones, and much triple phosphate; no sporules of sugar fungus detected, and no phosphate of lime; the urine is now rather offensive, its colour was slightly deepened by boiling with potash; but the copper test gave no evidence of sugar.

Examined 21*st Sept.* Much urate in globules at bottom of glass, with triple of phosphate, but no phosphate of lime.

9th SAMPLE:—Passed *7th September*, at bedtime, nearly neutral; specific gravity, 1016.

Examined 10*th September*. Very thick, alkaline; scum on surface composed of vibriones, and triple phosphate, deposit formed of same; no sporules of sugar fungus detected, and no phosphate of lime.

10th SAMPLE:—Passed early on the *7th September*, decidedly acid; specific gravity 1016, clear pale straw colour; colour deepened by potash; copper test gave no evidence of sugar.

Examined 9*th September*. A little turbid, contains much vesical epithelium, but no oxalate of lime.

Examined 10*th Sept.* Very thick; masses of sporules near the surface, falling to the bottom when the glass is moved; a few bubbles of carbonic gas; phosphatic scum on surface; urine smells rather offensively, but is still a little acid.

Examined 11*th Sept.* Scum more decided, composed chiefly of phosphate of lime, with a little triple phosphate and many vibriones; still faintly acid; bulky deposit formed of mucus; the small sporules of the sugar fungus and crystals of phosphate of lime.

Examined 16*th Sept.* Urine alkaline; no further development of the saccharine fungus.

Although the colour of most of the above urines was deepened by boiling with potash, yet this increase was not greater than is commonly observed in urines which do not contain sugar. It is usually stated that non-saccharine urine is bleached by boiling with potash. This is incorrect, as shown by me in a paper published in the 'Lancet,' March, 1851.[1] It almost invariably darkens every variety

[1] On the Tests for Sugar in the Urine, 'Lancet,' 1851.

of urine. Thus, in not one of the above samples did either the potash or copper tests betray the presence of sugar.

As scarcely any data exist tending to elucidate the early, and therefore the most important stages of diabetes, it may be useful to give a somewhat detailed description of the symptoms, by which for the last three or four years the case of incipient diabetes referred to was characterised.

Case.—William F—, æt. 35, of delicate organisation and nervous temperament, but free from organic disease, has for some years suffered considerably from chronic indigestion, as evidenced by frequently recurring attacks of headache and sickness; these were brought on by very slight causes, as any little error or excess in diet; the headaches were particularly distressing, and attended by giddiness and confusion of thought; the vomitings set in some hours after the commencement of the headaches, when these were unusually severe, and occurred as often as once or twice a week; each attack consisted of several successive fits of retching, and sometimes lasted as many as 10 or 12 hours, continuing long after the stomach had been well emptied. Within the last 8 months the headaches and sickness have nearly ceased, but occur still occasionally on any departure from the very temperate and regular method of living usually pursued.

The attacks were worst in summer, milder and less frequent in winter. For the last four or five years the patient has noticed that he passed his urine more frequently and in larger quantity than ordinary, his kidneys acting quickly on the slightest cause, as almost immediately after taking liquids of any kind, especially beer. The characters of the urine have already been described above. He has long also experienced a constant feeling of debility and exhaustion, both bodily and mental; as the appetite was generally good, he was unable to account for this extreme debility, and expressed a conviction that the large quantity of urine eliminated acted as a drain upon his system, the food and drink taken, by stimulating the kidneys, appearing rather to increase the exhaustion than to afford support.

Amongst the peculiar symptoms were the following:

1st. An occasional dry hacking cough without expectoration, and unaccompanied by symptoms of cold; this used to come on about noon, and was attended by slight febrile excitement; it was always removed for the time by food. 2d. Frequent dryness of the lips without positive thirst; this symptom attracted attention long previous to the discovery of sugar in the urine, and excited the suspicion that the case might possibly be one of incipient diabetes. 3d. Very great susceptibility to changes of temperature and weather; while rain, or the least dampness of the atmosphere, produced considerable depression; the heat of the sun seemed to inflame the blood, and to induce in it a state of fermentation.

The above symptoms, prior to the detection of the sugar, were set down to indigestion and the phosphatic condition of the urine; it is now clear, however, that they were mainly attributable to the sugar, the presence of which in the urine shows that the primary assimilative functions were very greatly at fault.

The patient attributed his bad health to excessive mental labour and long-continued anxiety. For some weeks past he has relaxed somewhat from study, has taken more exercise, the diet has been regulated, meat being allowed twice a day; as the result of all which, the health has considerably amended.

We have then occurring in the urine in different and very opposite states, two distinct species of fungus, the one being characteristic, to some extent, of the presence of albumen, and the other of sugar; but since the conditions necessary for the development of Penicilium glaucum all exist in saccharine urine, the only difference being the superaddition of sugar, we have next to ascertain whether the two species do not sometimes occur together in the same urine.

As might almost have been anticipated, the result of observation on this point is, that they not unfrequently occur together.

When the amount of sugar present is but small, the two fungi go on developing themselves almost in equal ratio,

each presenting its own distinctive characters, so that when they have attained their full development, part of the surface of the urine will be occupied with patches of the true saccharine torula, and part with those of Penicilium glaucum. In some cases, even the same tufts may be formed of the two species combined. (Pl. III, fig. 4.)

When, however, the quantity of sugar is considerable, the saccharine torula is developed with such rapidity and in such quantity as to outstrip the other species; and it is only when the fermentation has nearly come to an end, that Penicilium glaucum comes into view, and proceeds in its development.

We have, in the next place, to consider very briefly the chemical changes which ensue in saccharine urine placed under the three conditions already described, viz. free exposure to the air, partial exposure, and complete exclusion.

In all the specimens of saccharine urine freely exposed to the air, the following changes have ensued: the sugar has disappeared, carbonic acid has been evolved, and alcohol formed; of the alcohol part escapes into the air, diffusing a vinous odour, and part is converted into acetic acid. As the conversion of the sugar proceeds, the specific gravity of the urine becomes greatly lessened.

In the specimens partially exposed to the atmosphere, the urine, after a time, presented a gelatinous appearance, possessed a smell like sour milk, and was strongly acid; on analysis it was ascertained that the sugar had disappeared, that a small quantity of alcohol was present, and that the acidity was due to an abundance of acetic acid. In one or two of the samples, large quantities of oxalic acid in combination with lime were detected. As the saccharine fungus was imperfectly developed only, it is probable that in this case the greater part of the sugar passed directly into acetic acid.

In the specimens from which the air was excluded, as there was only a slight development of the sugar fungus, so was there scarcely any formation of alcohol; nevertheless, the sugar disappeared, and it was found on analysis to have

become converted into lactic acid, a small quantity of butyric acid, and what appeared to be aldehyde, from its smell and property of slightly reducing the oxide of silver, and giving a brownish-yellow coloration with liquor potassæ.

These several transformations of sugar are interesting, if not important; it has usually been considered that saccharine urine, when kept for any length of time, always undergoes the vinous fermentation; the lactic, acetic, butyric, and oxalic acids sometimes formed, as well as the circumstances which determine their formation, having been in general overlooked.

From a review, then, of the whole of the facts and observations above described, relating to the development of torulæ in saccharine urine, the following conclusions may be deduced:—

1st. That there is developed in saccharine urine, freely exposed to the air, a distinct species of fungus, which occurs in no other condition of that excretion.

2d. That this fungus is identical with the yeast plant.

3d. That it passes through three stages of development, any one of which is distinctive of the species.

4th. That since it is sometimes developed in urine in cases in which the potash and copper-tests fail to detect the presence of sugar, and in which, therefore, the quantity of sugar is not very considerable, it affords a most valuable and important test, and furnishes us with the means of detecting diabetes, even in its earliest stages.

5th. That the conditions necessary for its development are—free exposure to the air, an acid liquid, nitrogenous matter to act as a ferment, grape sugar or glucose, and a moderate temperature.

6th. That it may be developed at will in any sample of urine which is sufficiently acid, by the addition of a few grains of grape sugar.

7th. That when specimens of saccharine urine are imper-

fectly exposed to the air, the development of the fungus is incomplete only.

8th. That when the atmosphere is entirely excluded, no development of the fungus occurs.

9th. That in some few cases, where the quantity of sugar is very small, the fungus will cease to grow after having passed through the first stage only of its development, in consequence of the sugar, all having become converted into alcohol and carbonic acid.

10th. That sugar may be present in some very rare cases in small quantity, and yet the torulæ fail to be fully developed in consequence of the urine not possessing the necessary degree of acidity.

11th. That in such cases it is probable the development might be ensured by the addition of small quantities of phosphoric acid, or of a solution containing carbonic acid.

12th. That the presence of this fungus indicates the vinous fermentation, its development being accompanied by the disengagement of carbonic acid and the formation of alcohol.

13th. That the power of the fungus in aiding the transformation of the sugar is limited to the period when it is in the condition of sporules or yeast, the thallus and aërial fructification exerting no influence over the change.

14th. That in those cases in which the fungus is only partially developed, in consequence of imperfect exposure to the atmosphere, the sugar is converted chiefly into acetic acid, but a portion sometimes, also, into oxalic acid.

15th. That where the fungus is not developed at all, in consequence of the exclusion of the atmosphere, the sugar is transformed into lactic, acetic, and butyric acids, and also probably aldehyde.

16th. That since, in saccharine urine, the conditions requisite for the development of Penicilium glaucum exist, that species is likewise frequently met with in such urine.

17th. That in very many of the specimens of urine obtained from the patient labouring under diabetes, in a mild form, large quantities of crystallised phosphate of lime were detected.

POSTSCRIPT, *received January 11th*, 1853.—Since my paper on the Development of Torulæ in the Urine was read before the Royal Medical and Chirurgical Society, I have been so fortunate as to meet with another case, in which the urine threw down, on being allowed to stand for some time, an abundance of crystals of phosphate of lime, and which also contained some sugar, as shown by the development of the sugar fungus. This case affords, therefore, further and strong proof of the relation which I suggested might possibly be found to exist between crystals of phosphate of lime in the urine, and small quantities of sugar. The particulars of the case are as follows:—

CASE.—Mrs. T—, aged 32, of delicate constitution and nervous susceptible temperament, subject to dyspepsia, married, and has three children, the youngest only three months old. Attention was directed to the urine in consequence of the intense pain experienced on ceasing to micturate. The only sample of urine examined, and for which I am indebted to my brother, Dr. Hassall, of Richmond, was passed on the 19th of November; it was of specific gravity 1031, very acid, contained a large quantity of some pink urate, very many crystals of uric acid, and some octohædra of oxalate of lime; there was no albumen, nor could sugar be detected by means of Trommer's test.

Examined *22d November*.—The urine was still acid, and a cloud of vaginal epithelium had fallen to the bottom of the glass.

Examined *27th November*.—It was still acid, and the surface was covered all over with circular patches of Penicilium glaucum in the state of sporules. Under the microscope there was discovered, mixed up with the sporules, an abundance of crystals of phosphate of lime.

Examined *7th January*.—Urine alkaline; there was a scum of Penicilium in perfect fructification covering the whole surface of the urine, and mixed up with this were

immense numbers of globules of the urate and crystals of phosphate of lime, triple-phosphate, and oxalate of lime, together with some sporules and thallus of the sugar fungus.

POSTSCRIPT, *received August 27th*, 1853.—Since the above communication was presented to the Royal Medical and Chirurgical Society, I have on several occasions met with crystals of phosphate of lime in connection with the saccharine torula. The fact, therefore, that some close relation exists between these crystals and sugar in the urine may now be considered to be fully established.

NOTE.—It may be well to state that the credit of establishing the real nature of yeast is due to Cogniard-Latour. In 1835 and 1836 he communicated to the *Société Philomathique* some researches on Ferments, which were afterwards published in a journal called 'L'Institut.' In 1837 he presented to the Academy of Sciences his "Memoire sur le Fermentation Vineuse," which was published in the 68th volume of the 'Annales de Chemie et de Physique,' 1838.

SEQUEL TO A CASE

OF

ALBUMINOUS AND FATTY URINE,

PUBLISHED IN THE 'MEDICO-CHIRURGICAL TRANSACTIONS' FOR 1850,

WITH SOME ACCOUNT OF TWO OTHER CASES OF SO-CALLED CHYLOUS URINE.

BY

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Received Nov. 19th.—Read Dec. 14th, 1852.

In the 'Medico-Chirurgical Transactions' for 1850, I have given a statement of the examination of the urine of a patient suffering from so-called chylous urine, from the 6th of November, 1849, to the end of the 14th of June, 1850. It ceased to be chylous on the 14th of February, 1850; and from the end of that day to the end of the 14th of June the urine was observed 625 consecutive times, and it was not once chylous.

From the 14th of June to the end of the 4th of October, 1850, the urine was passed 572 times consecutively. It was healthy, and the appearances need not be more particularly mentioned. The urine then was healthy from the 14th of February, 1850, to the end of the 4th of October, 1850. In these 232 days it was passed 1197 times.

First relapse on the 5th of October, 1850:—

Urine passed at	6.10	a.m.	was natural.
„	9	„	natural.
„	1	p.m., after breakfast,	slightly chylous.
„	9.15	„	clear.

6th October :—

„	7 a.m.	natural.
„	9.15 „	natural.
„	11 „	natural.
„	4 p.m.	slightly chylous.
„	6.10 „	still less chylous.
„	9.50 „	natural.

One dose of 20 grains of gallic acid was taken this day.

7th.—There was no chylous appearance. The gallic acid was taken until the 27th, when it was omitted. The diet remained unchanged.

From the beginning of the 7th to the end of the 25th of December the urine was passed 442 times consecutively. It was on every occasion healthy.

December 25th.—He dined in company, and drank some spirits.

26th.—Second relapse :—

Water passed at	1.35 a.m.	natural.
„	7.50 „	natural.
„	9.15 „	natural.
„	12.20 p.m.	slightly chylous.
„	1.20 „ dinner	clear, but rather pale.
„	3.20 „	more chylous than before.
„	4.30 „	rather more chylous; passed at my house, loaded with albumen.
„	6.20 „	little chylous; 20 grains of gallic acid taken.
„	8.30 „	not so chylous.

27th :—

„	4 a.m.	still less chylous.
„	7.45 „	natural.
„	9.15 „	natural; cloudy from urate of ammonia; 20 grains of gallic acid taken.
„	10.20 „	the same.
„	12.15 p.m.	the same; 20 grains of gallic acid taken.
„	4.15 „	clear and natural; 20 grains of gallic acid.
„	8 „	natural.
„	12.30 a.m.	natural.

Dec. 28th.—The urine was passed 8 times. It was natural in appearance, and contained no albumen. Two doses of gallic acid only were taken at 9 a.m. and 12.15. p.m.

29th.—Only one small dose of gallic acid was taken this morning.

Water passed at	8	a.m.,	natural.
„	9	„	natural.
„	1.20	p.m.,	natural.
„	6.10	„	natural.
„	9.30	„	rather chylous.

30th :—

„	6.35	a.m.,	slightly chylous.
„	9	„	less chylous.
„	12	„	more chylous.
„	4	p.m.,	more chylous.
„	6	„	more chylous.
„	10.15	„	not so chylous.

Three full doses of 20 grains of gallic acid each were taken this day. No change in the diet was made.

For the six following days—that is, to the 5th of January—45 grains of gallic acid were taken daily. The urine was passed 36 times. It was natural 24 times; chylous 12 times.

For the ten following days 60 grains of gallic acid were taken daily. The urine was passed 70 times. It was natural 41 times; chylous 29 times.

During these 16 days the urine was natural each time it was passed on the 2d, 3d, 8th, 13th, and 14th of January.

The 15th was the last of the ten days.

Urine passed at	6.40	a.m.,	natural.
„	8	„	natural.
„	12	p.m.,	natural.
„	4	„	rather chylous.
„	6	„	not so chylous.
„	8.20	„	still less chylous.
„	10	„	very slightly chylous.

16th. — The urine was natural, and on the 7th of

February it was still healthy. It was observed 133 times consecutively. Sixty grains of gallic acid were taken daily until the 20th of January. On that day, and until the 26th, 45 grains were taken; on the 26th, 10 grains only; on the 27th to the 7th of February inclusive, 5 grains only. Then it was stopped.

On the 8th of February to the 9th of September inclusive the urine was healthy. It was passed 1203 times consecutively. No medicine was taken.

September 10th.—Third relapse.

Urine passed at 7 a.m., natural.
„ 9.30 „ natural.
„ 12.50 p.m., natural.
„ 4 „ rather chylous.
„ 6.30 „ not so chylous.
„ 9.45 „ not so chylous.

This chylous appearance was attributed by the patient to an over-large meal of fat mutton. He had not taken any spirits or stimulant of any kind.

11th :—

Urine passed at 5 a.m., very slightly chylous.
„ 7.30 „ natural.
„ 9.20 „ natural.
„ 1 p.m., natural.
„ 5 „ natural.
„ 9.50 „ natural.

From this day until the 27th of September the urine remained natural. It was passed 82 times. I examined it on the 27th of September, when it was healthy.

Fourth relapse on the 28th of September. He got up well and took porridge for breakfast.

Water passed at 7.30 a.m., natural.
„ 9.40 „ natural, contained no albumen.
„ 1.52 p.m., rather chylous.
„ 5.15 „ rather more chylous.
„ 9 „ not so chylous.

September 29th :—

Water passed at	5.15	a.m.,	very slightly chylous.
,,	8.15	,,	clear, slight trace of albumen.
,,	9.20	,,	rather chylous; very albuminous.
,,	11.5	,,	more chylous; specific gravity 1020·6.
,,	1.15	p.m.,	very chylous.
,,	4.15	,,	very chylous.
,,	7.45	,,	rather less chylous.
,,	10.30	,,	rather less chylous; albuminous; specific gravity 1010.

One drachm of gallic acid was taken this day in divided doses.

30th :—

Water passed at	6.35	a.m.,	no albumen; very slightly chylous; pale; specific gravity 1007; acid.
,,	8.25	,,	clear; healthy colour; albuminous; specific gravity 1020·4.
,,	9.50	,,	slightly chylous; very albuminous.
,,	12.15	p.m.,	very chylous.
,,	3.33	,,	very chylous, with blood.
,,	10.30	,,	very chylous.

One drachm and 20 grains of gallic acid were taken this day.

He got up at 6.40 a.m.; breakfasted at 8.20 on toast, tea, bread and butter; mutton and bread for dinner. Tea at 5 p.m.

October 1st :—

Water passed at	6.30	a.m.	slightly cloudy; contained no albumen.
,,	6.50	,,	slightly cloudy from chyle; contained albumen.
,,	8.15	,,	rather more chylous; contained much albumen.
,,	10.30	,,	still more chylous; much albumen.
,,	1.5	p.m.,	less chylous; little albumen.
,,	2.45	,,	still less chylous; very little albumen.
,,	5	,,	rather more chylous; little albumen.
,,	6.30	,,	rather more chylous; rather more albumen.
,,	10.30	,,	more chylous; much albumen.

He got up at 6.30 p.m., but kept quiet, resting between 10.30 and 2.45. He took one drachm and a half of gallic acid during the day.

October 2d :—

Water passed at	6.15	a.m.,	clear; no albumen.
„	8.15	„	clear; little albumen.
„	11	„	rather chylous; much albumen.
„	1	p.m.,	rather less chylous; less albumen.
„	5	„	much more chylous; milky; much albumen.
„	9	„	much less chylous; very little albumen.

He rested between 11 a.m. and 1 p.m., and between 1 and 5 p.m. Two drachms of gallic acid were taken during the day.

3d :—

Water passed at	6.25	a.m.,	clear; no albumen.
„	8.15	„	clear; little albumen.
„	12.30	p.m.,	slightly cloudy from chyle; no albumen.
„	4.10	„	nearly clear; no albumen.
„	7.10	„	more cloudy from chyle; no albumen.
„	9.30	„	less cloudy; no albumen.

4th :—

Water passed at	6	a.m.,	clear;	healthy colour;	no albumen.
„	8.15	„	clear	„	no albumen.
„	8.45	„	clear	„	no albumen.
„	10.10	„	clear	„	no albumen.
„	1	p.m.,	clear	„	no albumen.
„	3.10	„	clear	„	no albumen.
„	7.15	„	clear	„	no albumen.
„	9.30	„	clear	„	no albumen.
„	12.30	„	clear	„	no albumen.

Got up at 8 a.m., and by 9 a.m. had taken one drachm and 20 grains of gallic acid.

5th :—

Water passed at	7	a.m.,	clear; no albumen.
„	9	„	clear; no albumen.
„	10.20	„	clear; no albumen.

Water passed at 1.30 p.m., slightly chylous; much albumen.
„ 5.15 „ more chylous; much albumen.
„ 7.6 „ very chylous; very much albumen.
„ 10.45 „ very slightly chylous; very little albumen.

No gallic acid at all was taken from the morning of the 4th to 4 p.m. on the 5th. Then 40 grains were taken. The morning of the 6th, 80 grains of gallic acid were taken; and in the afternoon 40 grains more were taken.

October 6th :—

Water passed at 6 a.m., clear; no albumen.
„ 8.30 „ clear; no albumen.
„ 1 p.m., chylous; very albuminous.
„ 4.45 „ chylous; very albuminous.
„ 7.45 „ very chylous; very albuminous.
„ 10.50 „ very chylous; very albuminous.

7th :—

Water passed at 4.45 a.m., slightly cloudy from chyle; very slightly albuminous.
„ 6 „ clear; no albumen.
„ 8.45 „ very slightly cloudy from chyle: much albumen.
„ 10.10 „ slightly cloudy; much albumen.
„ 5.45 „ very chylous; very albuminous.

Two drachms of gallic acid were taken in the course of the day.

From this day to the 10th of November the gallic acid was continued in doses of two drachms daily. On the 31st of October the urine was slightly cloudy once. On the 1st of November it was perfectly healthy. All the other days it was more or less cloudy from chyle. The urine was passed 218 times. It was free from chyle 58 times; chylous in different degrees 160 times.

On the 10th of November the gallic acid was omitted and tannic acid was given, the first day only 20 grains. The four following days 60 grains. And then for seven days 90 grains. In all nearly 15 drachms in 12 days. The urine was passed 69 times in the 12 days. It was clear 6 times only; more or less chylous 63 times. The patient com-

plained much of the medicine, saying it caused much uneasiness of the stomach, and much pain in the head. He was exceedingly unwilling to continue it.

Solution of acetate of lead in acetic acid was then tried, two grains three times each day. It had no effect on the urine, which was passed 32 times. It was clear 4 times; more or less chylous 28 times. The acetate of lead was taken only five days, being omitted on the 27th of October, as he complained of pain in the bowels.

November 27th.—Nitrate of silver was tried in 2-grain doses three times each day. The pills were continued for four days.

The urine was passed 22 times. It was clear, natural twice, chylous in different degrees 20 times.

December 1st.—The gallic acid was resumed; two drachms daily, divided into three doses.

No remarkable change occurred, though the same dose of medicine was continued until—

January 22d, 1852.—The urine from December 1st up to this day was passed 175 times. It was clear 21 times; more or less chylous, 154 times.

This day at	7	a.m.,	very slightly cloudy with chyle.
„	9	„	rather loaded with chyle.
„	12	„	rather chylous.
„	4	„	very chylous, with a deposit of blood.
„	10	„	brown blood.
„	10.15	„	red blood: plenty of blood-globules.
„	11.15	„	few blood-globules.
„	11.30	„	clear, watery.

23d:—

„	7	a.m.,	clear, natural.
„	9	„	rather loaded with chyle.
„	12	„	much loaded with chyle.
„	5.20	„	rather chylous, with a brown deposit.
„	8.30	„	rather chylous, with blood.
„	10.15	„	rather chylous, with blood.

January 24th:—

Water passed at	7	a.m.,	rather loaded with chyle.
„	9	„	much loaded with blood and brown deposit.
„	9.30	„	slightly loaded, with blood.
„	10.15	„	clear and more natural.
„	12	„	very slightly loaded with chyle.
„	3.50	p.m.,	very slightly cloudy with chyle.
„	8	„	slightly chylous.
„	10.15	„	again less chylous.

Two drachms of gallic acid were still continued, divided into three doses.

25th:—

Water passed at	7.30	a.m.,	clear and natural.
„	9.20	„	same.
„	2.40	p.m.,	very slightly loaded with chyle.
„	7	„	the same.
„	10.15	„	clear, but rather pale.

26th:—

Water passed at	7.15	a.m.,	clear and natural.
„	9.20	„	the same, but rather pale.
„	12	„	the same.
„	2.30	p.m.,	very slightly loaded with chyle.
„	7	„	the same.
„	10	„	rather more loaded with chyle.

27th:—

Water passed at	1.15	a.m.,	clear, but rather pale.
„	7	„	clear and more natural.
„	9	„	the same.
„	12.30	p.m.,	very slightly cloudy, with chyle.
„	2.45	„	rather more cloudy, with chyle.
„	7	„	still more chylous.
„	9.30	„	not so chylous.

28th:—

Water passed at	7	a.m.,	clear, but rather pale.
„	9.50	„	clear and natural.
„	12.30	p.m.,	very slightly cloudy, with chyle.
„	4	„	rather chylous.
„	7	„	not so chylous.
„	10.15	„	still less chylous.

January 29th:—

Water passed at	5.30	a.m.,	clear, but rather pale.
„	8	„	clear, but natural.

Water passed at	9.15	a.m.,	clear, but rather pale.
„	12	„	very slightly cloudy, with chyle.
„	4	p.m.,	rather more cloudy.
„	7.30	„	much more chylous than any since 4 p.m., January 22.
„	10	„	much less chylous.

30th:—

Water passed at	7.10	a.m.,	clear, but rather pale.
„	10	„	clear, but natural.
„	12.30	p.m.,	very slightly cloudy, with chyle.
„	4	„	rather more cloudy.
„	7	„	less cloudy.
„	10.10	„	the same.

31st:—

Water passed at	5.45	a.m.,	very slightly cloudy, with chyle.
„	9	„	clear and natural.
„	12.15	p.m.,	very slightly cloudy, with chyle.
„	4	„	the same.
„	8	„	rather more cloudy.
„	10	„	clear and natural.

Thus this fourth relapse lasted from the 28th of September, 1851.

On February 1st, the water was free from chyle or albumen. The gallic acid was continued in 2-drachm doses until the 10th, when it was reduced to 1 drachm daily. On the 17th of February only 20 grains of gallic acid were taken daily. On the 1st of March 15 grains only were taken. This quantity was continued until the 3d of April.

From the 1st of February to the 29th of September the urine was passed 1290 times consecutively, It was not once chylous. On the 30th of September he sailed for the West Indies.[1]

The original paper, and this sequel together, constitute an abstract of the uninterrupted daily record of the state of the urine each time that it has been passed for nearly three years, from the 6th of November, 1849, to the 30th of September, 1852.

[1] He remained away seven months. At the end of this time I again saw him, and he told me the urine had remained perfectly free from chyle; and it continued healthy on the 20th of July, 1853. From the 1st of October, 1852, to the 20th of July, 1853, it was passed 1503 times.

The original paper appeared strongly in favour of the efficacy of gallic acid in the treatment of this disorder. The urine had been chylous for upwards of one year; and then, after the gallic acid had been taken for two days, it became healthy, and remained so for 232 days, the gallic acid being continued nearly two months. The urine was passed 1197 times free from any chylous appearance.

After this, as is seen in this sequel, the first relapse occurred. For two days a little albumen and fatty matter appeared in the urine. Twenty grains of gallic acid were taken, after which the urine remained healthy for 80 days, from the 7th of October to the 25th of December, 1850. The gallic acid was not omitted until the 27th of October, by which time 7 drachms more had been taken.

The second relapse lasted for 21 days, from the 26th of December to the 15th of January, 1851, inclusive. The urine was chylous for 15 days; free from chyle 6 days. During this time about two and a half ounces of gallic acid were taken. The urine then became healthy, and remained so for 237 days; that is, from the 16th of January to the 9th of September, inclusive. The gallic acid was continued from the 16th of January to the 7th of February, during which time $9\frac{1}{2}$ drachms of gallic acid were taken.

The third relapse lasted only two days. No gallic acid was taken. After this for 16 days (that is, from the 12th to the 27th of September, inclusive,) the urine was healthy.

Then the fourth and most serious relapse occurred. It lasted 34 days, from the 28th of September to the 31st of October, during which time 2 drachms of gallic acid were taken daily. On the 1st of November the urine was healthy. The next day it was again chylous; and, as it continued so on the 10th, tannic acid was tried in doses of a drachm and a drachm and a half daily. This acid was continued for 12 days with no success. Then acetate of lead was tried for 5 days; after this nitrate of silver was given for 4 days. These remedies failing, gallic acid in two drachms daily was again tried. It was taken from December 1st to January 31st, 1852. The urine was chylous every

day. So that this 4th relapse lasted 126 days, from the 28th of September, 1851, to the 31st of January, 1852, during which time about twenty-five ounces and a half of gallic acid, and nearly two ounces of tannic acid, were taken.

From the 1st of February the urine became healthy, and it was so on the 3d of April; that is, for 63 days. The gallic acid was continued in the following doses:—for 10 days, two drachms daily; for 6 days, one drachm daily; then 20 grains; and lastly, 15 grains daily. Thus nearly four ounces and seven drachms more were taken. In all, above 30 ounces of gallic acid were taken during this relapse.

From the 3d of April to the 29th of September; that is, for 179 days, the urine remained free from any appearance of fatty matter.[1]

The following table gives the duration of the chylous urine, and of the healthy state:

After the original attack had existed more than ten months, the

Urine was found to be chylous for	101 days.
It then became healthy, after gallic acid, for	232 „
Then, in the 1st relapse, the urine was chylous for	2 „
It then became healthy, after gallic acid, for	80 „
Then, in the 2d relapse, the urine was 6 days clear, chylous for	15 „
It then became healthy, after gallic acid, for	237 „
Then, in the 3d relapse, the urine was chylous for	2 „
It then became healthy after no gallic acid for	16 „
Then, in the 4th relapse, the urine was 1 day clear, chylous for	125 „
It then became healthy, after gallic acid, for[1]	242 „

During and after the original attack the quantity of

gallic acid taken was about			5 ounces		
„ „	1st relapse	„ „		7	drachms.
„ „	2d relapse	„ „	3 „	$5\frac{1}{2}$	„
„ „	3d relapse	„ „	none.		
„ „	4th relapse	„ „	30 „	3	„

Total amount of gallic acid taken, nearly 40 ounces.

[1] It continued healthy at the end of 292 days more—from the 1st of October to the 20th of July.

This enormous amount of gallic acid is sufficient evidence that it is no specific for so-called chylous urine. Its beneficial action as an astringent is, however, sufficiently evident, although it by no means acted so well and so quickly as it did when it was first prescribed.

It is interesting to mark the difference between the action of the gallic and tannic acids, the latter causing much more nausea than the former, and much more headache. Much larger doses of gallic acid could be taken than of tannic acid. Generally the dose of the latter should be one fourth or sixth of the former acid.

The effect of rest and exercise is also very evident throughout the sequel of this case, but this has been sufficiently dwelt on in the original paper.

The following case also illustrates the action of gallic and tannic acids, and shows the effect of rest and exercise on the appearance of the so-called chyle in the urine.

A gentleman, æt. 40, married, having four children, consulted me on the 11th of May, 1852. He said that he was born in Bermuda, and had passed his life in the West Indies, except during six years, when at different periods he came to England.

In 1842, when in Bermuda, he first found that the urine was milky. He had had the yellow fever four years previously, and had taken much calomel. He attributes the state of the urine to heat and cold. This milkiness of the urine lasted eight weeks, and went away without treatment.

In 1847, the disease returned. He passed clots, and semi-solid masses with some difficulty. The attack lasted two or three months. Alum did some good. The attack had almost subsided when he came to England, and consulted Dr. Prout, who in May, 1848, said that there was not the most remote sign of the complaint. He remained in England, well, until November, 1849.

In June, 1851, in Jamaica, the disease returned, and has continued ever since, with the exception of an interval of three weeks at the end of March and beginning of April,

1852. He left the West Indies in the middle of January, 1852.

Now the urine is cloudy; clears with æther; contains much albumen; some blood-globules; no fibrinous casts; is acid. Specific gravity 1025.

He thinks that bodily or mental exertion, even preaching on a Sunday, produces the most white water. Usually, the urine is thick on going to bed. Clear in the morning until an hour after breakfast, the whiteness then increases according to the exercise or exertion which he takes. He dines at 1, and then with rest the urine is clear, until he takes his afternoon walk, when the whiteness returns. He has tried all kinds of tonics, buchu, and iron. He has taken gallic acid, on this and the previous occasion, without advantage.

There was slight roughness with the first sound of the heart over the base towards the left clavicle. He has no pain in the loins. The bowels are regular, and the appetite very good.

. May 12, water passed 4 a.m.; specific gravity 1018·5, contained the slightest trace of albumen.

„ „ 8 „ specific gravity 1021·0, no albumen, by heat or acid.

„ „ 11 „ urine whiter than usual, attributes this to exercise.

Acidi Gallici, 3j, quotidie; Ext. Colocynth. comp. Pil. Rhei, comp. āā gr. ijss, hora somni.

	Morning.	Noon.	Afternoon.	Bedtime.
May 13, water passed,	clear	turbid	turbid	turbid
„ 14 „	„	„	„	thick
„ 15 „	„	thick	thick	„
„ 16 „	„	„	white	„
„ 17 „	„	„	„	„
„ 18 „	„	white	„	„
„ 19 „	„	„	„	white

During the week, the drachm of gallic acid was taken in three portions. The diet was nutritious, without stimulants. The exercise was very little. The urine became more white,

[1] By thick is meant that the urine *when passed* is of a dirty whitish-brown appearance, and untransparent.

with pain and uneasiness about the loins. The bowels became very confined, never acting without the pill; a drachm and a half of gallic acid each day was prescribed.

		Morning.	Noon.	Afternoon.	Bedtime.
May 20,	water passed,	clear	white	white	white
,, 21	,,	,,	turbid	turbid	thick
,, 22	,,	,,	,,	thick	white
,, 23	,,	,,	white	,,	thick
,, 24	,,	,,	turbid	white	,,
,, 25	,,	,,	clear	clear	clear
,, 26	,,	,,	,,	,,	,,

In consequence of headache during the last two days, great quietness was observed, the patient remaining in an easy recumbent position. Two drachms of gallic acid to be taken daily.

		Morning.	Noon.	Afternoon.	Bedtime.
May 27,	water passed,	clear	clear	clear	clear
,, 28	,,	,,	,,	,,	,,
,, 29	,,	,,	,,	,,	,,
,, 30	,,	,,	white	white	thick
,, 31	,,	,,	turbid	,,	,,

The improvement in the urine continued until the 30th, when on returning from church, it was found quite white, "showing thereby its close connection with fatigue." The forenoon of the 31st, it was also turbid.

		Morning.	Noon.	Afternoon.	Bedtime.
June 1,	water passed,	clear	turbid	white	white
,, 2	,,	,,	thick	,,	,,
,, 3	,,	,,	,,	,,	thick
,, 4	,,	,,	turbid	,,	white
,, 5	,,	,,	thick	,,	,,
,, 6	,,	,,	,,	,,	,,
,, 7	,,	,,	,,	,,	,,
,, 8	,,	,,	,,	turbid	,,
,, 9	,,	,,	turbid	white	,,
,, 10	,,	,,	thick	,,	thick
,, 11	,,	,,	turbid	,,	,,
,, 12	,,	,,	,,	turbid	,,
,, 13	,,	,,	white	,,	turbid
,, 14	,,	,,	clear	clear	clear

		Morning.	Noon.	Afternoon.	Bedtime.
June 15,	water passed,	clear	clear	clear	clear
„ 16	„	„	„	„	thick
„ 17	„	„	„	„	clear
„ 18	„	„	„	„	„
„ 19	„	„	thick	thick	thick
„ 20	„	„	white	white	white

The urine became clear on the 14th, but in consequence of a journey to London, or some other cause, it became cloudy on the 19th. The gallic acid was continued in two drachms daily up to the 15th, when it was omitted, and has not been resumed since. The bowels have been very confined, and there has been some giddiness of the head, not amounting to headache. Has had a little nausea. To resume the gallic acid in two drachms daily.

		Morning.	Noon.	Afternoon.	Bedtime.
June 21,	water passed,	clear	thick	white	white
„ 22	„	„	„	„	„
„ 23	„	„	„	„	„
„ 24	„	„	„	„	„
„ 25	„	„	turbid	„	„
„ 26	„	„	„	„	„
„ 27	„	„	thick	„	„
„ 28	„	„	turbid	„	„
„ 29	„	„	„	„	„
„ 30	„	„	thick	„	„
July 1	„	„	clear	clear	clear
„ 2	„	„	„	„	„
„ 3	„	„	„	„	„
„ 4	„	„	„	„	„
„ 5	„	„	„	„	„
„ 6	„	„	„	white	thick
„ 7	„	„	turbid	thick	„
„ 8	„	„	thick	white	white
„ 9	„	„	„	„	„
„ 10	{ 3 drachms of gal. acid daily }	„	„	„	„
„ 11	„	„	„	„	„
„ 12	„	„	„	„	turbid
„ 13	„	„	„	„	thick
„ 14	„	„	„	„	white
„ 15	„	„	clear	thick	„
„ 16	„	clear	thick	white	white

		Morning.	Noon.	Afternoon.	Bedtime.
July 17,	water passed,	„	„	„	„
„ 18	„	„	„	„	„
„ 19	„	„	„	„	„
„ 20	„	„	„	„	„
„ 21	„	„	„	„	„
„ 22	„	„	„	thick	turbid
„ 23	„	„	turbid	turbid	„
„ 24	„	„	„	white	white
„ 25	„	„	„	„	„
„ 26	„	„	„	„	„
„ 27	„	„	„	„	„
„ 28	„	„	thick	„	„
„ 29	„	„	„	„	„
„ 30	„	„	„	„	„
„ 31	„	„	„	„	„
Aug. 1 to 22	when gallic acid stopped	„	„	„	„
„ 23	tannic acid begun, one drachm daily	„	thick	white	white
„ 24	„	„	clear	clear	clear
„ 25, can only take half a drachm daily, the full quantity caused painful sickness of the stomach and very uncomfortable feelings.					
	water passed,	clear	clear	clear	clear
„ 26	„	„	„	„	„
„ 27	„	„	„	„	„
„ 28	„	„	„	„	„
„ 29	„	„	„	„	„
„ 30	„	„	„	„	„
„ 31	„	„	„	„	„
Sept. 1 to 8	„	„	„	„	„

9th.—My patient wrote to me, "The tannic acid makes me uncomfortable, by producing a sensation of nausea immediately after swallowing it, and I am compelled to lie on my back in order to retain it on my stomach. So violent was the vomiting produced by the third of a full dose (20 grains), that I was obliged to reduce the quantity to one half (10 grains). I cannot retain it when taken on an empty stomach, either in a large or small dose. The day after I commenced it the urine became clear, and with the exception of being turbid at midday on Sunday, the 5th,

has continued clear. The gallic acid only produced an unpleasant taste in the mouth, which soon went off. The action of the bowels was rather more free with the gallic acid."

	Morning.	Noon.	Afternoon.	Bedtime.
Sept. 10 to 22, water passed,	clear	clear	clear	clear.

September 22d.—"The urine has continued clear, with the exception of Sunday the 12th and yesterday the 21st, when there were interruptions of a few hours each. On the 12th, I was very quiet at church; on the 21st, I had taken a little more exercise than usual. The medicine is as nauseous as ever."

	Morning.	Noon.	Afternoon.	Bedtime.
Oct. 10, water passed,	clear	clear	clear	clear
„ 11 „	whitish	whitish	whitish	whitish
„ 12 „	„	„	„	„
„ 13 „	„	„	„	„

The quantity of tannin was increased again to 30 grains, which "is as much as I can retain on my stomach without sickness."

	Morning.	Noon.	Afternoon.	Bedtime.
Oct. 14, water passed,	clear	whitish	whitish	whitish
„ 15 „	„	„	„	„
„ 16 „	„	„	„	„
„ 17 „	„	„	„	„
„ 18 „	„	„	„	„
„ 19 „	„	„	„	„

The tannic acid was left off, and the gallic acid was taken in drachm doses, three times, daily.

Oct. 25th.—Not the slightest change produced in the appearance of the urine. The pain in the back very distressing.

	Morning.	Noon.	Afternoon.	Bedtime.
Oct. 26, water passed,	clear	whitish	whitish	whitish
„ 27 „	„	„	„	rather clearer
„ 28 „	„	„	„	„
„ 29 „	„	„	„	„
„ 30 „	„	clear	clear	thick
„ 31 „	„	very thick	thick	„

November 1st.—The gallic acid was left off, and instead, tannate of alumina, with very dilute sulphuric acid, was ordered. This was soon changed for tannin, with sulphate of alumina.

		Morning.	Noon.	Afternoon.	Bedtime.
Nov.	1, the urine was	clear	thick[1]	thick	thick
„	2	„	„	„	„
„	3	„	„	„	„
„	4	„	„	„	„
„	5	„	„	not quite clear	not quite clear
„	6	„	„	„	„
„	7	„	„	„	„
„	8	„	„	rather thick	„
„	9	„	clear	clear	clear
„	10	„	„	„	„

On the 19th of February, 1853, the medicine was diminished, and in a fortnight more it was altogether omitted. The urine remained perfectly free from chyle, and it continued so up to the time of his last visit to me, on the 10th of August.

The following case shows that this disease is not necessarily serious. It has lasted, off and on, more or less for a quarter of a century, and has caused so little inconvenience, that the patient is unwilling to try any remedy for its cure.

In the third edition of Dr. Prout's work on 'Stomach and Urinary Disease,' 1840, p. 116, writing on chylo-serous urine, he says: "In one case which I attended, and in which acute inflammation of the liver, with phlogistic fever, took place, the urine during the continuance of the acute symptoms lost entirely its chylous character. The same remarkable disappearance of the chylous symptoms also subsequently occurred in the same patient during the presence of severe ptyalism from mercury. But as soon as the inflammatory action and the mercurial excitement had ceased, the chylous condition of the urine returned even worse than before." At page 115 the same case I have every reason to believe is mentioned. Dr. Prout says: "I had an opportunity of examining the urine passed in the morning and after dinner within a few weeks after the disease in its worse form had been apparently arrested by medical treatment, and to my surprise I found both specimens perfectly free from albuminous matter, and, as far as

[1] "Thick, means less transparent than turbid, and of a more dirty colour. It does not become thicker on cooling."

I could determine, quite natural." In a note to the fifth edition of 1848 he says the lady whose case is here alluded to, a native of the East Indies, and in whom the disease first occurred twenty-one years ago, was recently alive and in good health.

I was requested by Dr. Mac Loughlin to see this lady on the 3d of December, 1851. Her age was then 57, and she weighed 14 stone.

She told me that the urine became chylous in 1827; that it remained so for two years; and that she considers it became clear from bathing; that it remained perfectly healthy for 13 years, part of which time she was in India; that the white appearance returned after bleeding and salivation in 1843; that it remained until 1845, when, after strong mental emotion, it disappeared, and was absent six months. It returned in consequence of over-exercise, and it has remained ever since. No clear water at all has been seen since 1846, excepting once, when she thinks the water was made clear for one night in consequence of some opium which she took.

I saw the urine which was passed during the night, after breakfast, after dinner, and after exercise. It was quite milky, and contained some clots, which I was told occasionally appear as if moulded into the form of the urinary passage.

A good deal of pain in the back over the region of the kidneys was complained of. Otherwise, the health was very good, and exercise in walking was generally taken for two hours each day. No medicine whatever was taken, and the patient has ceased to expect that any change will occur in the state of the urine.

I advised the trial of gallic acid in ten given doses two or three times a day. She took only one dose; said that it caused some unpleasant feelings in the head and tingling in the fingers; and she refused to take any more gallic acid.

I again visited this lady in February, 1853, and I examined the urine, which was still chylous; and in July Dr. Mac Loughlin informed me that she remained in the same state as at the time of my visit.

ON

DEGENERATION OF THE PLACENTA

AT THE END OF PREGNANCY.

BY

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COMMUNICATED BY

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THE diseases of the Placenta have formed the subject of several elaborate essays. Degeneration of that organ, likewise, as a presumed cause of abortion, has been brought under the notice of the profession in this country by Dr. Robert Barnes. But there are some questions relating to this degeneration which require to be answered before its real value, as a cause of disease, can be determined. In the first place, it must be considered how far, and under what circumstances, it may be regarded as a normal condition—how frequently, and to what extent, it may be present without any ill consequences ensuing. In the second place, our knowledge of the diseases of the placenta will require to be revised, in order to ascertain to what extent mere degeneration may heretofore have been mistaken for disease. The following observations on the former of these questions are laid before the Royal Medical and Chirurgical Society, in the hope of exciting the attention of the Society and of the profession to the subject, which is not destitute of important relations to practice.

By degeneration, it is almost unnecessary to say, is meant the loss of those characteristic parts, or marks of any given structure, which are most intimately connected with its functions. Such a change bears witness, as Professor Paget has remarked, rather to a deficiency than to a perversion of nutrition. There is no development of any new organic form; but, instead, a tendency to infiltration with oil globules, or with earthy matter. In structures whose existence is shorter than that of the system of which they form a part, such a change is met with towards the close of their existence. In organs which have occasional periods of activity, such a change is met with at the end of those periods. Thus, degeneration is to be looked upon as a part of the natural, as well as of the morbid, history of the animal œconomy.

Now, the placenta, being *par excellence* a temporary organ, might fairly be expected to be prone to degenerate towards the close of its term of office; and this I believe to be true, and that almost every placenta expelled at the full term exhibits some signs of that change.

It is, in fact, notorious that various deviations from perfect structure are constantly found in placentæ, although in favorable cases, if noticed at all, they are passed over as of no consequence, whilst in unfavorable cases, in which the placenta is apt to be scrutinised far more nicely, they are usually attributed to the effects of inflammation and effusion of lymph. This error, I confess, I have constantly committed, until the above-mentioned paper by Dr. Barnes revealed the true nature of many of these alterations. But I soon became convinced that degeneration of the placenta, to a certain degree, was anything but an uncommon condition; and I determined, when at leisure, to institute a more strict inquiry, the results of which I now lay before the Society.

But, to prevent confusion, let me say one word on the terms employed, since eminent [illegible]gists are not yet quite agreed on the names to h[illegible] certain parts of the placental structu[illegible]ct that loops of

fœtal vessels, sheathed in processes of chorion, are implanted into a venous sponge, whose covering is the decidua. Thus, the fœtal are separated from the maternal vessels by two membranes—the decidua nearer to the maternal, the chorion to the fœtal. On looking at a fœtal villus, it is perceived to be bounded by a clear edge, which represents the lining of the maternal venous system. Within this, and closely adherent, is an investing layer of beautiful nucleated cells, which Dr. Hassall calls *chorion*, but which, in accordance with the views of Professor Goodsir, supported by Professor Quekett, and confirmed by observation of the analogous parts in animals, I assume to be *decidual*. When this investing layer is stripped off, the chorion, containing the fœtal vessels, comes into view.

The cells of the decidua, at the end of pregnancy, are found in three conditions. First, there are those which I have been speaking of as the decidual cells investing the fœtal vessels. These are believed to be the active functional parts of the placenta, and to have the office of extracting from the maternal blood the matters which are absorbed by the fœtal. Secondly, there are those cells which have been developed into fibrous tissue, to form the requisite mechanical elements of the placenta. And, thirdly, other cells found in variable abundance, on the maternal surface, between the lobules, under the chorion, and along the track of the larger fœtal vessels. These are found in every variety, rounded and elongated, and partially split into fibres, and, I presume, are to be looked upon as a residue of surplus material, which has not been brought into active functional use, because it has not been in contact with the minute villi of the chorion, and which has not been required for purposes merely mechanical. These cells are called in the following observations *free decidual cells*.

These things being premised, the forms of degeneration commonly seen in the placenta are the earthy and the fatty.

The earthy degeneration is so common, that I doubt if any full-grown placenta is to be met with that does not

show some traces of it. It seems to begin in the terminal tufts of the lobules, and usually on the uterine surface; but it may pervade the entire substance of various lobules, rendering them of almost stony hardness. When the affected tissue is examined microscopically, the first traces of earthy deposit are seen in the form of minute transparent crystals, within the investing decidual cells of the fœtal villi. Their presence is soon attended with shrinking of the villi, and decay of the definite cellular structure of their envelope. As the crystals accumulate, they completely involve and bury the smaller tufts, which look like the twigs of a tree laden with snow, and then they creep along the larger branches. The effect of the accumulation of earthy matter must be evident. On the one hand, the vessels compressed and separated from their envelope, become atrophied; and, on the other, the cells are torn up; and when the crystals are dissolved out, appear confused and ragged; or else stripped off from the chorion.

This earthy matter is quickly and entirely soluble in acetic acid, with copious effervescence. This circumstance distinguishes the crystals from oil globules, which the earliest deposits strikingly resemble, although they do not float out into the liquid under the field of the microscope, as the oil globules do. The quantity of earth in the placenta is often immense, constituting nearly the whole weight of the affected lobules when dried, and representing a proportionate quantity of placental tissue quite incapacitated for performing its functions. Dr. Lionel S. Beale, who was good enough to analyse it for me, found it to consist of the phosphate and carbonate of lime, with a trace of magnesia.

The fatty degeneration is as common as the earthy, and they are generally met with in the same placentæ, though not in the same parts, or in equal degrees. It was most fully and accurately described by Dr. Barnes and Dr. Hassall, but I will venture to add a few details.

As regards that part of the placenta which I have termed the free decidual cells, it begins with the presence of, first,

single globules, then clusters in the cells; and at last they and the fibrous tissue about them are completely gorged with oil, and resemble to the naked eye a soft slimy yellowish fat. This is to be found in variable abundance at the edge of almost every placenta, where it is intermixed with the atrophied remains of the vessels which once ramified in the membranes. It is often found also covering the maternal surface of many of the lobules, as well as between them, and under the chorion on the fœtal surface. In any situation it may contain a few healthy tufts carrying blood, and several others shrunken and atrophied. This it is which constitutes the fat spoken of by Wilde in his 'Treatise on Diseases of the Placenta,' as so often found on the surfaces of the organ; and likewise, I believe, the substance which William Hunter speaks of, as resembling the sizy layer of pleuritic blood at the edge of the placenta. But that which looks like fat or lymph to the naked eye, consists merely of fibrous tissue, and degenerate decidual cells, gorged with oil. This is spoken of in the following memorandum as *loose fatty stuff*.

In the working part of the placenta, the fatty degeneration begins in like manner with the deposit, first of globules, then of clusters of them, into the investing decidual cells. As it increases, in one class of cases, the tissues become more and more oily and soft, till there is left a mere soft slimy mass, in which, however, the course of the decayed vessels can be made out by careful manipulation and maceration in acetic acid.

In other cases, the oil does not increase in proportion to the degree of degeneration; in fact, neither the oil globules nor the earthy crystals are to be looked upon as more than accidents, and not as constituting the essence of the degeneration. But be this as it may, the affected tufts become, as truly described by Drs. Barnes and Hassall, tallowy, ex-sanguine, brittle, and difficult to unravel; whilst in the extreme stage, there is produced a white, glistening, translucent, amorphous substance, of gristly consistence, and breaking in all directions into sharp angular fragments.

But in this apparently structureless substance careful examination, aided by maceration in water, enables us to detect the forms of the fœtal vessels, which crop out here and there on the broken surfaces; and of which, mixed with condensed free decidual cells, the whole of it consists. In this extreme degree of degeneration there is very little oil. Acetic acid causes this substance instantly to swell up, and become translucent.

The evidence on which the foregoing statement is based, is contained in the following memorandum of the examination of placentæ occurring consecutively in my own practice and that of a neighbour. Each placenta was examined whilst quite fresh, and every part of it scrutinised most carefully, so as to ascertain the real nature of the healthy and morbid structure contained. In some placentæ many lobules, coated with soft fatty decidual cells, were quite perfect within; whilst in other instances, lobules that were sound externally, had their central portions in a complete state of fatty degeneration.

Memoranda of Placentæ examined.

1. Patient æt. 32, first child; had severe rheumatic pain and tenderness of uterus three weeks before delivery; labour natural; placenta, a very well-marked partial ring of hard fatty degeneration.

2. Patient æt. 27, sixth pregnancy, including one abortion; in the interval between the former pregnancy and this, had suffered severely from uterine congestion and leucorrhœa; twice during this pregnancy had slight sanguineous discharge, lasting a week each time; labour natural; placenta, slight margin of loose fat, but on the whole very free from degeneration.

3. Patient æt. 26, third pregnancy; labour at 270th day (so far as the patient could reckon), from the commencement of last menstruation; parturition very painful; irregular uterine contraction and hæmorrhage after birth of child; placenta extracted by hand; it was quite entire; had several white gristly lobules in state of hard fatty degeneration.

4. Patient æt. 26, first pregnancy; confined on 269th day from beginning of last menstruation; dated the beginning of labour from a sudden fright four days previous to it; child small; placenta expelled entire, without assistance; several lobules completely degenerate in fatty and fibrous masses: sent it to King's College Museum to be injected and sketched by Dr. Westmacott.

5. Patient æt. 28, first pregnancy; labour tedious; child large; placenta, several fatty lobules near margin.

6. Patient æt. 24, first pregnancy; labour natural; placenta free from degeneration, except a few earthy specks.

7. Patient æt. 21, first pregnancy; had slight sanguineous discharge for one night, about the sixth week; labour at 289th day; child, a girl, weighed $8\frac{1}{2}$ lbs.; placenta, drained of blood, $1\frac{1}{2}$ lb.; a very large lobule in state of complete fatty and fibrous degeneration.

8. Patient æt. 28, sixth pregnancy, including one abortion; labour at 280th day; very tedious from early rupture of membranes; placenta very free from degeneration, except a few earthy specks.

9. A lobule in very well-marked fatty degeneration, from a patient of Dr. Peregrine's.

10. Patient æt. 25, first pregnancy; labour tedious; delivery at 268th day after marriage, and 271st from beginning of last catamenia; child, $5\frac{1}{2}$ lbs.; placenta small, no degeneration, but plenty of loose fatty stuff.

11. Patient æt. 25, first pregnancy; labour natural at 268th day from marriage, and 286th from commencement of last catamenia; child large; placenta, two or three lobules gristly from fibrous degeneration. A portion, which displayed other changes, sent to Dr. Lionel Beale, who thus reports on it:

"This portion contained a firm whitish substance about the size of a pea, adhering intimately to the surrounding textures, and splitting into thin laminæ when attempts were made to remove it. It very much resembled coagulated fibrine in its microscopical appearances. The tufts at the

margin of this placenta appeared for the most part healthy, but a few were dark and granular; others presented a fibrous appearance, whilst, in a few, oil globules were noticed, but were not so abundant as in the other specimens submitted to examination."

12. This, and the following twelve specimens, occurred consecutively in the practice of a neighbour:

Much loose fat, and at the edge all varieties of degeneration, from slight shrinking and accumulation of oil, to complete brittleness and obliteration of all natural structure. One large vein filled with laminated fibrine. A portion of this placenta was thus reported on by Dr. Beale:—

"In the midst of the spongy tissue was a hard and almost gristly spot, about the size of a small pea. A portion of this was examined by the microscope. For the most part it appeared to be composed of a glistening material, which here and there had a distinctly fibrous structure, and in some places small collections of fat globules were observed. This substance broke up, rather than tore, into sharp angular fragments, many of which were quite transparent, and had no appearance of fibrous structure. When subjected to pressure they gave way, but on the removal of the pressure they regained their former shape. A few small and shrivelled tufts were here and there observed; some had a fibrous appearance, and others contained collections of oil globules and granular matter. No cellular structure could be detected in any of them. Upon the addition of acetic acid, the whole became more transparent, the fat globules more distinct, and in the tufts a few nuclei could be detected. All appearance of fibrous structure had at the same time vanished."

13. This specimen contained much fatty and fibrous degeneration at the margin and between the lobules.

14. Much earthy deposit, and several patches exhibiting every gradation, from healthy tissue to amorphous gristle. Fig. 5, by Dr. Beale, gives the appearance of tufts in an advanced stage of oily degeneration.

15. Full of earthy deposit: very little fat.

16. Some loose fatty stuff; one or two marginal lobules quite atrophied. Fig. 10 shows a sketch of withered tufts extracted from the edge of this placenta. A portion was sent to Dr. Beale, who reports thus:

"Numerous small whitish particles were observed in the substance of the mass. A few of these, unravelled with needles, appeared to consist entirely of tufts which had originally been collected very closely together. Some, however, were found to have a simple fibrous appearance, the fibres being arranged concentrically, and resembling very closely small portions of certain fibrous tumours. Acetic acid caused the development of nucleus-like spots, which were also arranged in a concentric form. The tufts of this portion of the placenta appeared perfectly healthy, and the cells very clear and distinct, with the exception of those situated quite at the margin of the placenta, which were small and shrunken, exhibiting more or less opacity; some presenting a fibrous structure, whilst others contained collections of fat and separate oil globules in considerable abundance. The tissue in the intervals of the tufts was also loaded with oil, as has been observed in other specimens submitted to examination."

17. A few calcified specks; very little other degeneration.

18. One of twin placentæ, contained some atrophied tufts in loose fat. In one lobule, examined by Dr. Beale, "was a whitish lump, the size of a pea, of gristly consistence, giving, when cut, the sensation of fibro-cartilage, not readily torn, but breaking into small fragments when attempts were made to unravel it with needles. On examining some of these fragments, they were found to consist for the most part of a glistening material, exhibiting here and there an indistinctly fibrous structure, with a little granular matter, and a few small oil globules. In some places a few small tufts were seen; they had a somewhat glistening appearance, and occasionally a few ill-developed cells were observed. In other tufts numerous deposits of small oil

globules; and in some of these, the oily deposit occupied the whole diameter of the tuft, and extended some distance along it."

19. The corresponding placenta showed much earth, and little else.

20. A very definite gristly edge, and very much earthy deposit; besides a remarkable atrophied marginal lobule, worthy of separate description. On the fœtal surface, under the amnion, was an oblong patch of what looked precisely like a layer of fibrine. It was found to consist of a marginal lobule quite atrophied; the investing chorion doubled up; it was seen on a section to be filled with a yellowish oily cobwebby substance, composed of the remains of excessively atrophied vessels, reduced almost to a diffluent mass. On applying acetic acid, lines of vessels, which had lost all cohesion, were rendered visible.

21. Very little fat, but much earth; one lobule of almost stony hardness.

22. Very little fat; but an immense quantity of earth in the lobules and on their surfaces.

23. Much loose fat, some earth, and one lobule in a state of complete fibrous degeneration in the centre.

24. Patient æt. 29, fifth pregnancy; labour natural; placenta very large, and looking like a model of healthy structure; yet one lobule at the centre and one at the edge had in the middle of each a most perfect example of fatty degeneration in the advanced stage.

25. A few earthy specks, one very small fatty patch, and plenty of loose fat.

26. Some soft fatty degeneration at the edge; several of the lobules had incipient fatty or fibrous degeneration in their centres; one large vein in the centre filled with dirty brown fibrine.

27. The fœtal surface near the margin had a raised edge of what looked like lymph, but which was in fact hard atrophied tissue, as described by Dr. Beale in No. 11; there was besides much soft fat.

28. Patient in advanced stage of phthisis; very little

degeneration, except a few earthy specks, and a little loose fatty stuff at the edge.

29. A great quantity of loose fatty stuff, filled with fœtal vessels in state of oily and earthy degeneration.

30. Patient æt. 26; a remarkably fine woman; second pregnancy; infant very small; born at 285th day from beginning of last catamenia; infant expelled with two pains as patient was standing; placenta very small, thin, tough, and leathery; elastic, like one that has been immersed in alcohol; many small lobules at one edge, converted into white lumps. The tough portion, on microscopical examination, was condensed and brittle; investing decidual cells, very easily stripping off from the vessels, and universally glittering with minute points of oil. The white lumps composed partly of stuff like white laminated fibrine, partly of atrophied tufts, and healthy ones in dense fibrous tissue. Each white lump had a large venous channel through it.

The foregoing memorandum confirmed me in the impression I had previously formed—that traces of fatty or earthy degeneration are to be found in all mature human placentæ, and noticeable degrees of it in at least a fourth. In the above cases there were two appearances which are possibly morbid. I allude to the fibrinous tumours in Nos. 11 and 12. The pseudo-morbid appearances were very common. That in No. 20 has been noticed; but in most cases some of the loose fatty stuff might easily have been mistaken for coagulated fibrine. In several instances the chorion was thickened, even to half an inch, and was easily divisible into layers, and infibrated with oil globules; in others some of the lobules were softened, and slightly broken down in the centre, and filled with dark coagula (constituting the so-called apoplexy). These appearances I notice, although they are pseudo-morbid and trivial. I have used the term human placentæ, because, although I am told on the best authority that earthy degeneration is very frequent in the secundines of cattle, I am unable to verify the statement from my own observation. I have

found in the secundines of a calf plenty of oil in the decidual cells, but only one small cotyledon out of 50 or 60 of all sizes that could really be called degenerate.

In the foregoing cases the alterations in the placenta were quite irrespective of any circumstances in the age and health of the mother, the duration of the labour, the separation of the placenta, or the welfare of the child. The mass of calcified placenta may be so large (as in a case lately related to me by Dr. Griffith) as to injure the passages of the mother in its extraction; but there is no sufficient evidence that either this, or any other form of degeneration, causes, mechanically or otherwise, either premature separation of the placenta before birth or adhesion of it afterwards.

The true explanation of the phenomenon, I humbly conceive, must be looked for in the general analogies which regulate the growth of temporary organs. The placenta is the nutrient organ of a parasite, which inhabits its parent until it has attained a certain degree of development. Its cells, like other cells in a condition of active growth, have the power of appropriating nourishment from any parts with which they are in contact—whether from the uterus, as in normal pregnancy, or from the ovary, fallopian tube, or peritonæum, in abnormal instances. It is probable that the size and condition of the placenta bear a strict proportion to the wants of the fœtus and to its powers of assimilation; and that, on the one hand, a placenta of perfect structure shows that the functions of the organ are actively carried on; and, on the other hand, that degeneration shows either that the organ was originally formed on an unnecessarily large scale, or that the fœtus cannot appropriate the supplies furnished by the entire organ, or that, the work of development being nearly completed, the active employment of the whole organ is unnecessary, and portions fall into decay.

This view of the physiological nature of degeneration of the placenta is confirmed by the pathological history of that condition, as in the cases published by Dr. Barnes, Dr. Handfield Jones, and others. A pregnant woman is

exposed to circumstances which cause the death of the fœtus, quickly or slowly. But the blighted ovum is not immediately expelled, and the placenta, no longer employed, undergoes degeneration.

It is the physical condition of the cells of the chorion, and of the blood in the fœtal vessels, which is the determining cause of the changes which go on in the decidual structures, just as the presence of an hydatid causes changes in whatever parts it infests. But when the physical condition of the fœtus is such that it cannot imbibe the supplies conveyed to it by its blood from the decidual cells, then that blood loses *ipso facto* the condition through which it imbibes those supplies, and the decidual cells become first gorged, then stagnant, then loaded with inorganic products, lastly degenerate.

On a future occasion, I hope to lay before the Society some remarks on the degree in which the induration, hepatization, scirrhus, ossification, chondrosis, and steatoma, spoken of by authors as affecting the placenta, may be resolved into some form of degeneration. At present I will merely add three conclusions which I have ventured to draw, viz.:

1. That incipient degeneration is a normal condition of the placenta at the end of pregnancy.

2. That it arises from partial cessation of the active functions of the organ, when the fœtal development is nearly completed.

3. That, when occurring in the earlier months, it *probably* arises from some antecedent want of nutritive force in the fœtus, or by its death.

Lastly, with regard to the proposition of inducing premature labour, on the ground of placental disease, I would say, in the words of Wilde: "Nostrum est, summo studio cavere, ne abortus expediatur vel immo excitetur; sed omni arte potius intendere ut prospera et immunis restituatur graviditas; quippe quâ unâ, duplicis vitæ lætam spem et sinceram salutem recuperavimus."[1]

[1] De placentæ morbis, p. 34.

EXPLANATION OF THE PLATES.

Figs. 5, 6, 7, 8, show the comparative condition of similar parts from an ovum of about seven weeks, and from the secundines at full term. 5. A group of villi from the non-placental portion of the chorion of an early ovum; most of them quite healthy; but in some few, a little oil is visible. 6. Shows the villi from the non-placental portion of the chorion, at the end of pregnancy.

Many writers have overlooked the fact of the persistence of these villi to the end of gestation, and have speculated on their absorption. But they are to be found on every part of the chorion, down to the very part which is ruptured to allow the passage of the fœtus. In the placenta itself, many villi may often be found, under the chorion, which seem to have never received blood-vessels into them. Such villi may be found in abundance at the edge of every placenta; and if the decidua be stripped from the chorion, they may be found at intervals in great abundance all over the surface of that membrane. They evidently have continued to increase in length, though not in number, during the earlier months of pregnancy. At the end of the term, they are found flattened out, imbedded in the oily decidua which covers them; they often contain a good many oil globules; are often ampullated or bulged, as if in a state of incipient cystic disease; and their extremities are usually doubled back.

Fig. 7, shows decidual cells from the same ovum; and 8, and others, from a mature placenta. I have never examined any placenta from which I could not procure specimens of oily decidua, similar to that depicted in Dr. Hassall's very elaborate and beautiful drawing in the 'Med.-Chir. Trans.,' 1851, rendering it very doubtful in my mind to what extent oily deposit in the fœtal membranes is to be regarded as morbid.

HYPERTROPHY AND PROLAPSE OF THE TONGUE.

BY

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Margaret Kelly, æt. 11, was sent from the Isle of Man, by Dr. Kemp, to be treated in Addenbrooke's Hospital, May, 1847. I learnt from the mother that when the child was between two and three years old she had sore throat and hooping-cough; that powders were then given, which made the mouth sore and the tongue swollen; that during a fit of coughing the swollen tongue protruded from the mouth, and remained so. She recovered her health, but the tongue continued to increase in size up to the time of her admission into the hospital.

At that time the enlarged tongue (pl. I)[1] hung from the mouth over the chin in front of the neck, measuring from the upper lip to its tip $3\frac{1}{2}$ inches; from the under lip to its tip $1\frac{1}{2}$ inch; from the angle of the mouth, round the sides and tip to the opposite angle, $6\frac{1}{4}$ inches. The circumference of the widest part, which was about the middle of the protruded portion, measured $6\frac{1}{2}$ inches circular measurement;

[1] Taken from a drawing, for which I am indebted to my friend Mr. Bailey, surgeon, Cambridge.

immediately within the lips, 5 inches. When drawn into the mouth to the utmost possible extent the tongue measured from the upper lip to its tip 2 inches. The organ was soft and supple, having been kept habitually covered in a bag slung from the head. It presented no ulceration or apparent traces of inflammatory or other disease, except simple hypertrophy. The papillæ were greatly enlarged and separated by deep clefts, giving to the exterior of the mass a coarsely granular or warty appearance. This appearance was not confined to the prolapsing portion; it extended also to the part of the tongue contained within the mouth, though in a somewhat less degree. The colour of the organ was natural, or nearly so. The opening of the mouth was large: the lower lip everted, and the angles of the mouth depressed, so as to elongate and give a peculiar expression to the face. The orifices of the sublingual ducts, of unusual size, were situated just in front of the edge of the lip. The saliva was continually dripping from the end of the tongue; the quantity thus lost amounting, during the day, to more than half a pint.

Owing to the constant pressure of the tongue on the mental portion of the lower jaw, a curvature had taken place in that bone, just in front of the masseter muscles, in such a manner that a wide interval (pl. II) always existed between the incisors and bicuspids of the two jaws. Even when the mouth was closed, that is to say when the corresponding molar teeth were in contact, this interval between the incisors measured nearly two inches, being increased by the horizontal direction which the inferior incisors and the alveolar process of the lower jaw had assumed. These were so placed as to form a wide channel in which the tongue rested. Moreover, the teeth, especially the two central incisors, were farther apart than natural, and encrusted with tartar, which in some measure filled up the spaces between them, and prevented their sharp edges from injuriously pressing upon the tongue. In consequence of this altered configuration of the jaw, and the horizontal position of the front teeth, sufficient space was left

for the tongue to hang out of the mouth, although the superior and inferior molars were in contact. Even mastication with these hinder teeth could be effected without any injury or compression of the protruding organ.

The os hyoides retained nearly its natural position with relation to the jaw, and there was nothing remarkable in the throat or neck. The gums were swollen and spongy.

The child could masticate and swallow with tolerable ease. She bit soft substances between the upper lip and the tongue, and by the movements of the latter and of the lips contrived to roll the morsel about the mouth. Her speech was very thick and indistinct, though intelligible. She suffered no pain, but could not bear to be seen, and was exceedingly anxious to be rid of her deformity.

It being clear that the removal of the prolapsing portion offered the most expeditious and certain, if not the only effectual, mode of relieving the child from her distressing malady, and the knife offering many advantages over the ligature, I passed a straight bistoury from below upwards, through the organ a little to the left of the mesial line, and cutting forwards and outwards formed a left lateral flap; secured a vessel; next I cut across the middle of the tongue, dividing the ranine arteries, the movements of the organ being still controlled by means of the prolapsing portion, which was not yet quite severed. The operation was completed by the formation of an oblique lateral flap on the right side, corresponding with that on the left. These flaps were approximated so as to form a tip, and maintained in apposition by two sutures passed deeply. The execution of these proceedings was, of course, much facilitated by the wide interval that existed between the incisor teeth. The hæmorrhage was not very great, and the bleeding vessels were tied without difficulty.

A good deal of swelling of the tongue followed the operation; but under frequent washing and fomentation it soon subsided, and the wound healed. At first the lips could not be approximated, and the thick stump of tongue was always visible, though never protruding. After a few days, however, by

the continued action of the orbicularis muscle, the mouth was closed. I carefully cleared away the tartar from the incisor teeth, and placed between them and the lower lip small pads of linen to prevent the mucous membrane of the latter suffering from pressure against their sharp edges. It was not long before they assumed their natural direction. The jaw also began to recover its proper shape. To expedite the latter process I had a cap made of stout calico and metal, with a hooked bar of iron projecting from it, like a horn, over the forehead. This bar was attached to the hinder part of the frame-work of the cap by a hinge, and to the forepart by a screw, which enabled me to alter its elevation according to circumstances. A thick belt of India-rubber passed from the hook underneath the chin and exerted constant pressure upon it. The apparatus was worn for several hours at a time, and doubtless contributed to bring about the improvement which took place. When its use was commenced on January 18th, four months after the operation, the interval between the maxillary alveoli was $1\frac{5}{8}$ inch, having decreased about a quarter of an inch. On February 22d it was $1\frac{1}{4}$ inch. In August $\frac{7}{8}$ inch. After this the change took place very slowly, though the deformity was at length almost removed. The remaining portion of the tongue underwent considerable reduction in size during the first few months after the operation; then became stationary. Great improvement took place in her speech. She returned to her home in September, 1849, having, for about a year previously, lived as servant in my house, at the latter part of which time the only defect observable by strangers was a slight thickness in her speech. A friend who visited her in the Island in the summer of 1852, found her quite well.

When I was a student at the Norwich Hospital, in the year 1837, a case corresponding with the above in almost every particular, except that the prolapsing organ was not quite so large, was treated by Mr. Crosse, who devoted much time and attention to the subject, collected informa-

tion from various quarters with his usual assiduity, and intended to have written a memoir upon it. The following is a brief outline of the case:[1]

Sarah Cole, æt. 6, from Dickleburgh, in Norfolk, had at birth, under the tongue, a small ranula, which was opened by a surgeon; afterwards the tongue enlarged. No treatment being adopted, the organ continued to increase in size, and ultimately hung out of the mouth. At the time of her admission into the hospital it measured from the upper lip to the tip, 3 inches; circular measurement, 5 inches. She could articulate intelligibly. The changes in the lower jaw and teeth, and the general appearance of the tongue, were the same as in the preceding case. Mr. Crosse at first contemplated removing the part by ligature, fearing hæmorrhage if the knife were used; but happening to see a paper by Lassus, he determined to try pressure, being influenced by the strong terms in which that treatment is there recommended, and the cases adduced in its favour. The application of a few leeches caused an increase in the swelling, and was not repeated.

October 5th.—A bandage, soaked in solution of alum, was placed round the tongue, so as to effect moderately firm compression. This was covered with oil-silk, and the whole enclosed in a sling attached to a linen cap fitted to the head. Liquid food was given through an elastic tube, so as to prevent the necessity of the removal of the bandages, which were renewed every morning. The child was laid on her back. After this treatment had been perseveringly continued for seventeen days, the tongue was so much reduced in size that it could without difficulty be replaced in the mouth: no part projected beyond the lips, though the latter could not be closed. The circular bandage was now discontinued, and the tongue kept in the mouth by a linen bandage passed over it.

November 12th.—The child could approximate the lips

[1] There is a cast of the face in the Pathological Museum of the University of Cambridge.

so as to close the mouth; the still-enlarged tongue caused bulging of the cheeks on either side.

January 3d, 1838.—The bandages were left off all day, and an elastic strap, passing under the chin and over the head, for the purpose of elevating the mental portion of the jaw, was worn instead. In consequence of the altered shape of the lower jaw, a constrained effort of the orbicularis muscle was still required to close the mouth, which gave to the oral aperture a circular shape and puckered outline.

February 15th.—She was made out-patient, with directions to continue the use of the chin-strap.

June 2d.—The child was re-admitted, with the tongue swollen, and prolapsing nearly as bad as ever; the relapse attributed to severe cold and cough. No improvement in the shape of the jaw since her first admission. The former treatment was pursued, and in five days the tongue was again in the mouth. The case went on favorably till May 15th, when the child became sick and feverish, and the tongue again protruded to the same extent as formerly. This attack subsided, and the tongue was a third time reduced within the confines of the lips. I am not aware of any subsequent relapse.

The child remained under Mr. Crosse's care till August, 1839, when the tongue was still large, though kept within the lips; the chin-strap was still worn; and a bandage was kept over the mouth at night. Some elevation of the mental portion of the jaw had taken place, and three permanent incisors had sprung up in a natural direction in place of the milk teeth, which had been extracted, or had fallen out. In this condition the child again went home, and I saw no more of her. A letter from Mr. Harrison, surgeon, Diss, dated July 8th, 1851, informed me that at that time the tongue remained perfectly in the mouth, although, when she spoke, it was perceptible slightly beyond the teeth. The articulation was thick, though distinct. The jaws appeared in a natural state.

My friend, Mr. Ormerod, told me of a similar case at

Oxford; and when I was in that city, at the meeting of the Provincial Medical and Surgical Association, not long ago, Mr. Hester kindly took me to see it. It corresponded in all important particulars with the two preceding; but the tongue was not proportionately so large. It projected from the mouth about $2\frac{1}{2}$ inches; was dry on the surface, but its substance was soft, and it presented no ulceration or sign of inflammatory disease. The lower lip was everted; the lower jaw bent, so that mastication could be effected only between the hinder teeth. The incisors had been extracted long ago. The woman (æt. 46) could speak quite intelligibly, only a little thick. Her mother had told her that the tongue was at birth of natural size; that a gathering took place when she was an infant, and the tongue swelled; it got better, but, in consequence of another gathering, it swelled again, and had remained out of the mouth ever since, being sometimes much larger than at others. A variety of applications had been tried, but no systematic treatment adopted. The poor woman was very anxious to be relieved from her malady, and ready to submit to any plan that offered a reasonable prospect of success.

The disease illustrated by these cases appears to be one of simple nature, consisting merely of hypertrophy of the tissues of the tongue. The amputated portion in that first described showed that nearly all the tissues had participated in the change. The mucous membrane in particular was much thickened, and thrown up into large, closely-crowded, branched processes, corresponding with the papillæ. It was not quite clear that the proper muscular structure itself had undergone any hypertrophy. The fibres of that tissue, presenting perfectly natural appearance under the microscope, were arranged in coarse bundles, rendered strong and tough by thick areolar and fibrous investments. These thickened structures were not interwoven quite so closely as in the healthy tongue, the interstices for blood-vessels and fluids being rather larger and more numerous than usual; but I could discover no trace of any morbid product.

In all three cases the affection was traceable to an inflammatory attack in the first instance. Subsequently, the unnatural and dependent position of the organ, the constriction by the lips, and other causes combined to promote its continued enlargement, and to prevent the spontaneous cure which commonly takes place when the tongue has swollen in ordinary glossitis.

I was induced to amputate the prolapsing organ, because I found that the operation had been performed in several instances with good result. Moreover, I had witnessed the tedious troublesome process necessary to effect decided improvement in the case treated at Norwich. Mr. Crosse, who came over to Cambridge to see my patient, quite agreed in the propriety of removing a portion of the tongue, and of removing it with the knife. Nevertheless, his case is a remarkable example of the benefit that may result from compression assiduously and perseveringly maintained. It proves that a cure may be thus effected even in a severe and long-standing case: *à fortiori* it quite confirms the observations of Lassus and others, that in the early stages of the disease compression is the proper, indeed the only, justifiable treatment.

It appears that but few instances of the disease have occurred in this country. Sir A. Cooper informed Mr. Crosse that he had treated a case exactly similar to the one under his care successfully by ligature. The patient was a girl, æt. 18. One of Mr. Syme's dressers, who examined the casts and drawings of my patient, told me that he had seen a similar case in the Royal Infirmary at Edinburgh not very long ago. The patient was a girl, æt. about 14; the enlarged tongue hung out of the mouth three inches. Mr. Syme removed the prolapsing part by oblique incisions, meeting at an angle in the centre of the tongue, and united the lateral flaps by sutures. The child died a few days after from inflammation and swelling of the tongue and the parts about the larynx. — Mr. Liston, in his 'Elements of Surgery,' relates a case of congenital enlargement of the tongue, similar in many respects to those I have described,

although the organ seems to have been more vascular, and the source of occasional severe hæmorrhages; the disease was regarded by him as of erectile character. The patient was a male, æt. 18. Mr. Liston interrupted its vascular supply by tying both lingual arteries. On the seventh day inflammatory swelling supervened, followed by sloughing. The protruded part was then isolated by ligature; the system became disordered; abscesses occurred in various parts of the body; and the patient died. These are the only two cases that have come to my knowledge in which the treatment by removal or other ways has been attended with a fatal result.

The affection (under the names of *lingua vituli, lingua propendula, macro-glossia,*) was described long ago by various authors.[1] The best account of it is by Lassus, whose

[1] Gaspar Peucer, in his commentary 'De Generibus Divinationum,' Witebergæ, 1572, p. 435, speaking of monsters, says: "Vidimus et qui exertas ac ceu dependentes tenuent linguas instar vitulorum recens jugulatorum."

Zacchias, 'Quæstiones Medico-Legales,' lib. vii, tit. 1, quæstio 9, relates that he saw at Rome, in 1628, a male infant, well made, except that the tongue projected three fingers' breadth from the mouth. The child could suck, and lived to the age of 14 months, when it died without obvious cause.

Bartholin, 'Hist. Centur.,' iii. p. 85, mentions a case of "linguæ putentosa magnitudo," related to him by his pupil Bogdun. A male child, born with the tongue out of the mouth as large as a filbert. As the child grew, the tongue increased to the size of a calf's heart, but did not prevent his masticating and speaking distinctly.

Sauvages, in his 'Nosologia,' speaks of enlargement of the tongue in new-born infants; also of enlargement arising from inflammation and other causes, but does not distinctly describe this disease.

The following case from Triven 'Observat. Med.,' p. 142, and Sandifort 'Obs. Anat. Path.,' lib. iv, cap. 9, is quoted by Lassus. A girl at Leyden had in her infancy continued fever, followed by swelling and protrusion of the tongue from the mouth. It hung out upon the chin four inches. At the age of 14, to conceal the deformity, she adopted the plan of enclosing the prolapsing organ in a silver box. She could speak and masticate. At the age of 54 she was seen by Sandifort. The tongue then filled the mouth, and hung out of it four inches and a half: its surface

description[1] has been copied by most subsequent writers. He opposes the practice of removing the prolapsing part, and strongly recommends the treatment by pressure, which he believes would prove successful even in the most inveterate cases. He observes, that the malady has never been known to undergo a spontaneous cure; but when the organ is returned into the mouth, and retained there, the swelling gradually subsides. He adopted this treatment with success in the case of a male infant eight days old, where the tongue projected from the mouth about a finger's breadth. Having stimulated the extremity with a little powdered alum, he replaced the organ in the mouth, and retained it there by means of a compress and bandage, which was removed only when the child took food. A radical cure was thus effected in 15 days. In this case the labour had been long and difficult; the face presented and was swollen, the mouth open, and the tongue protruded at the time of birth.—In another case, a lad, æt. 6 years, the tongue was enlarged and protruded three fingers' breadth; the deformity, of three years' duration, was attributed to a bad habit the child had contracted. The application of a few leeches diminished the volume of the tongue, and facilitated its return into the mouth, where it was retained by bandages, and a cure was effected in about a month.—A still more remarkable case related by him is that of a woman, æt. 40, who had for thirty years laboured under this disease. She was treated

uneven, tuberculated, covered with thick saliva. It caused continued heavy pain about the os hyoides. She was subject to frequent attacks of quinsy and difficulty of swallowing; but at other times could swallow easily, and speak with sufficient distinctness to be intelligible.

Galien 'De Diff. Morb.,' lib. i, cap. 9; Valescus, lib. ii, cap. 66; Alex. Benedictus, lib. v, cap. 2, 'De Cur. Morb.;' Scaliger, 'Exercitat.,' 199, cap. i; M. Donatus, 'Hist. Mirab.,' lib. vi, cap. 3; Martin Boydan; Paul de Sorbait; Bardet, 'Bulletin des Sciences Médicales de la Société d'Evreux,' No. 23, p. 67, are all quoted in the 'Dict. des Sciences Médicales,' as mentioning the disease, or as having seen cases.

[1] 'Mémoires de l'Institut National des Sciences—Mathematiques et Physiques,' tome i, 1799. A translation of this Memoir was published in the 'London Medical and Physical Journal,' 1801.

successfully by Louis, who enveloped the tongue in rags sodden with lettuce juice. After six months the tongue was returned into the mouth, and had remained there seven or eight years.

From the cases above mentioned, and those described by other writers,[1] all of which present great similarity in their

[1] In 'Dict. des Sciences Médicales,' vol. xxvii, is an article by Percy and Laurent, giving a full account of this disease, with two cases seen by Percy. The first, a woman, native of Petersbach, whose tongue, large at birth, had increased greatly in consequence of an inflammatory attack when she was three years old, and had retained about the same size from that period to the time when she was seen by Percy, her age then being 18. She declined to submit to the operation which was proposed. The second case, a lad, æt. 16, whose tongue hung 3 inches below the chin, was $2\frac{1}{2}$ inches thick, and filled the mouth. The affection had existed from birth: the teeth were displaced, and the lower jaw deformed. Percy removed the prolapsing portion with the knife, and a cure was quickly effected.

Boyer 'Traité des Maladies Chirurgicales,' tome v, gives a graphic account of the malady, and describes well the attendant changes in the lower jaw and mouth; says the tongue is covered with "mamelons," which may be also developed on the palate (these are doubtless the enlarged papillæ); observes that the affection is usually congenital, or occurring in early life, and that it may be due sometimes to paralysis of the retractor muscles of the tongue, but adduces no evidence or argument in support of the latter opinion. He quotes from the 'Mémoires de la Société de Médecine de Montpellier,' 1816, a case related by M. Mirault, surgeon at Angers. (This case is also related, and figured before and after the cure, in 'Dict. des Sciences Méd.') The patient, a man æt. 34, had suffered under the malady from infancy. M. Mirault removed the prolapsing portion, which weighed $3\frac{1}{2}$ ounces, by means of three ligatures. Failing, after a month's efforts, with the assistance of an expert dentist, to give to the displaced teeth their natural direction, he extracted them, and corrected the deformity and superabundance of the lower lip by removing a V-shaped portion.

M. Maurant 'Journal de Médecine,' vol. xv, gives the particulars of a lad, whose tongue hung from his mouth four fingers' breadth, and was 2 inches thick. He could masticate, and speak, and even sing. The tongue was large at the time of birth. No treatment adopted.

M. de Bierken 'London Medical Journal,' 1815, treated a girl, æt. 12, with tongue so large that it hung over her chin, causing eversion of the lip, &c.; deglutition was difficult; the voice unintelligible, and the

general features, we learn that the disease is one of sufficiently definite character to merit special notice. Though it sometimes originates in an inflammatory affection, it often

larynx projected. The disease had existed from the age of two years. It had been once cured temporarily by compression. Extirpation was resolved on, and effected by ligature, as in Mirault's case: a needle double threaded was passed through the tongue on either side of the middle line, the central and the two lateral portions being thus separately constricted. A cure was effected; and it is added that the teeth, after being extracted, were replaced and properly secured.

Dr. Wells, of Columbia, 'Medical Gazette,' vol. ii, had a patient, a girl, æt. 6, with tongue hanging over chin 2½ inches. The affection had commenced at eighteen months. Latterly, the growth had corresponded only with that of the girl. Prolapsing portion removed by ligatures placed obliquely, so as to give the remaining tongue a somewhat pointed shape. A good deal of irritation and constitutional disturbance followed; but in seven weeks the wound had healed, and the tongue became of natural size.

Dr. Harris, 'American Journal of Medical Sciences,' vol. xv, relates the case of a male, æt. 19. Tongue projecting beyond incisors 3 inches; circumference, 6 inches. Alteration in direction of teeth and jaw as usual. Affection congenital. Enlargement going on gradually. Dr. H. having dissected the tongue a little from floor of mouth, removed the protruding portion by two incisions meeting at an angle behind. The flaps approximated, and united by stitches. Sharp inflammation followed; but the wound healed in fourteen days. Some molar teeth were subsequently drawn to assist in approximating the incisiors. In fifteen months the deformity was removed, and articulation distinct. Dr. Harris published a similar case in vol. vii of same journal, except that the disease in that instance commenced at the age of 4 years.

'Med. Chir. Zeitung,' vol. xxxi. Amputation of prolapsing tongue, by Professor Leber, of Vienna. Girl, æt. 6 years; wound healed quickly.—Vol. lxix. Girl, æt. 10, born with long tongue. It protruded 4½ inches. Treated with ligature by Siebold. Case doing well, and wound healed, when the girl fell into a fever and died. A description, taken from post-mortem examination, is given of the alteration of jaw and teeth, corresponding with that in the cases I have related.—Lad, æt. 9; tongue hanging out of mouth 3 inches. Dr. Klein removed the protruding part with bistoury. Alarming hæmorrhage ensued, but was arrested. Next day tongue swollen to former size; the swelling subsided, and an improvement in the shape of the jaw gradually took place.—Vol. lxx. Prolapsing tongue removed by knife, by Rudtorrff.—Vol. 105. Tongue

appears to be independent of any such cause. It commences usually in childhood or early infancy, or is congenital. If it be early attended to, it admits of an easy cure by compression. If it be allowed to take its course,

protruded 5 inches; treated successively, by Dr. Edhold, with ligature in the same manner as the case by Bierken.—Vols. 111 and 116. Two, cases in which swelling and prolapse remaining after inflammation were in a short time reduced, and cures effected by pressure.—Vol. 148: Lad, æt. 5, tongue enlarged from birth; protruded 2¾ inches. Amputation by Dr. Newman, in Warren county. The wound soon healed, and the attendant deformities were subsequently rectified.

Gräfe und Walther 'Journal der Chirurgie.' A lad, æt. 8, after variety of treatment, the prolapsing portion, measuring nearly 3 inches, removed by the knife, by Dr. Nevermann. The bleeding controlled by actual cautery, and a cure effected.

'Archives Générales de Médicine,' 1844. Case from 'Giornale dei Progressi,' Juin, 1843. Male child, born with hypertrophy of tongue. The prolapsing portion, increasing with years, was removed by Dr. Federici. Two lateral flaps united by sutures. A cure quickly effected. —1838. From 'American Journal,' 1838. Lad, æt. 13, hypertrophy of tongue from birth. The organ had acquired enormous dimensions. Mr. Mussey removed the protruding portion in the same manner, as in preceding case. Cure rapid.—1842. From 'Casper's Wochenschrift,' 1842. Girl, æt. 6; tongue, enlarging three months, projected from mouth, and presented the usual appearances. Treated by Ruhbaum, of Rathenow, with lotions and mild aperients. A cure soon effected.

Louis, in a paper on Diseases of the Tongue, in 'Mém. de l'Academie de Chirurgie,' mentions the case of a man, whose tongue swelled during salivation, protruded from the mouth, and increased in size. The measures adopted having failed to reduce it, amputation was performed by Pimpernell. The wound healed, and the patient was able to speak, as before the occurrence of the disease. This severe treatment is reprobated by Louis, by M. Freteau of Nantes, who subsequently published in 'Recueil general de Médecine,' (see Medical Repository, vol. vii,) the case of a woman, æt. 24, whose tongue had been swollen for about six weeks without obvious cause, resisting treatment by incisions, and other means. Amputation was proposed; but M. Freteau effected a cure in a few days by pressure.

Dr. Clanny, of Bishop-Wearmouth, in 'Edin. Med. and Phys. Journal,' vol. i, gives the case of a lad, æt. 5. Tongue protruded 3 inches, began to swell when a year old. It was treated by pressure in accordance with the principles laid down by Lassus, and a cure soon effected.

the prolapsing organ increases; at first, in greater proportion than the growth of the child. After a time it would appear to become stationary, or to grow only with the growth of the body, for in none of the cases, even those in which the deformity had existed for many years, had the tongue attained to greater size than one's fist. Even in the most aggravated cases, a cure may be hoped from the persevering employment of pressure; but where the disease is of long continuance the process is necessarily tedious. Spontaneous cure rarely, if ever, takes place after the prolapse has been established. The operation of removal is not attended with much danger, and the remaining portion of the tongue shows no disposition to enlarge again, so that there is no reason to fear a return of the malady. Neither, on the other hand, do I find that the stump has in any case shrunk below its proper dimensions. Its size, provided a sufficient portion be left, which is ensured by removing so much only as prolapses, is regulated by the cavity of the mouth, and continues sufficient to fulfil the purposes of the organ.

The displacement of the lower incisor teeth and the altered configuration of the jaw, caused by the continual pressure of the heavy prolapsing organ, forms a curious and important feature in the disease, enabling the patient to masticate with the mouth open, and the tongue hanging out. I have seen the same change both in the teeth and jaw produced, in a less degree, by the contraction of a cicatrix after a burn on the front of the neck, dragging the chin down towards the sternum. It is a good illustration of the subserviency and adaptability of the skeleton to the influence and uses of the softer structures. In the cases we are considering, it greatly increases the difficulty of effecting a permanent cure by compression, because the tongue, deprived of its internal support, has a tendency to prolapse again from the mouth during sleep, and at other times; and the return of the teeth and jaw to their natural position is impeded by the pressure of the large tongue pent up within the confines of the lips, almost as much as when

it hangs out of the mouth. These points were sufficiently exemplified in the case of Sarah Cole. Of course the removal of the prolapsing portion greatly facilitates this part of the cure. I was surprised how quickly after the operation, in Margaret Kelly, the teeth became erect, and the mental portion of the jaw began to be raised under the combined influence of the pressure of the lips, the contraction of the orbicularis, and the circular elastic belt.

In the cases which I have been able to collect, the respective modes of treatment by pressure, ligature, and the knife were adopted, as follows:—

	Pressure.		*Ligature.*	*Amputation.*
	Duration of Disease.			
Lassus	8 days.		Cooper.	Percy.
„	3 years.		Liston.	Harris (2 cases).
Louis	30 years.		Mirault.	Leber.
Ruhbaum	3 months.		Bierken.	Klein.
Clanny	4 years.		Wells.	Rudtorrff.
Freteau	6 weeks.		Siebold.	Newman.
Crosse	6 years.		Edhold.	Nevermann.
				Federici.
				Mussey.
				Pimpernell.
				Syme.
				Humphry.

In Bierken's case the disease had been temporarily cured by pressure.

In all the above cases a cure was effected; except in Liston's and Syme's, where the treatment was attended with a fatal result. In Siebold's case, the girl died of fever during her recovery from the effects of the ligature, the slough having been detached, and the wound healed.

EXPLANATION OF THE PLATES.

Plate I

Shows the hypertrophied and protruded tongue, with its enlarged papillæ.

Plate II

Shows the curvature in the lower jaw, causing a wide interval between the upper and lower incisors, although the molars are in contact. A slight curve in the opposite direction in the alveoli of the upper jaw, which existed in the case, is rather too forcibly represented in the drawing. The teeth of the lower jaw are more widely separated than natural, and the gums between them swollen.

HYPERTROPHY OF THE TONGUE,

BEING

THE ACCOUNT OF A CASE OF THAT DISEASE, WHICH WAS SUCCESSFULLY TREATED BY LIGATURE.

BY

J. HODGSON, Esq., F.R.S.,

PRESIDENT.

Received Feb. 8th.—Read Feb. 8th, 1853.

A little girl, named Harriet Howes, æt. 2 years, was admitted into the Birmingham General Hospital on the 15th of April, 1833. The following is the report of her case, which was taken by my friend, Mr. Bowman, of King's College, at the time a resident student in the hospital:

The tongue, enormously enlarged, hangs out of the mouth, and reaches below the chin. It completely fills up the orifice of the mouth, and doubles down the under lip, which is always in contact with the chin. If the mouth be opened, it may be perceived that the part of the tongue within the lips does not present any unnatural appearance, either as to size or colour, except that the papillæ are whitish and slightly raised from the surface. Immediately on emerging from the mouth the tongue is enlarged, both in breadth and thickness, and there is an evident constriction where it has pressed against the teeth and lips. The greatest transverse extent of the projecting portion is $2\frac{3}{8}$ inches, and the length, anterior to the lips, rather more than 2 inches. The mother says that the child was born with a large tongue, and that when about two months old a leech was applied to the tip, which seems to have caused an increased action

in the blood-vessels, for, since that time, the organ has continued to increase in size. Mr. Hodgson first saw the child about twelve months ago; she was then in very bad health; the whole surface of the tongue was inflamed, excoriated, and extremely irritable. He did not then advise any operation, expecting that the child would not live many days; but her health improved, and she is now as strong as infants of her age generally are. The projecting portion of the tongue, however, exhibits vestiges of the inflammation. Externally to the mouth the papillæ are raised above the surface, and present the appearance of small pimples. The projecting part is of a deep red colour, dry and rough on the upper surface, where in places scabs or crusts have formed. The child could suck like other infants, and she can now eat and drink easily. She could talk when 16 months old, and now articulates nearly as well as children of her age. The chief inconvenience which she suffers is from the continual dribbling of saliva from her tongue and mouth. She can move the projecting part from side to side, or protrude it forward, and the muscular fibres appear to extend to its anterior extremity. The mother says that the tongue could be retracted into the mouth until the child was two months old, when the leech was applied, since which time it could not be even replaced within the lips. The anterior part of the lower jaw has been pressed out of its perpendicular, by the protruding growth, almost into a horizontal position, so that the lower incisor teeth project forwards, and their edges are not in the same line as those of the upper jaw.

On the 20th of April the protruding portion of the tongue was included in ligatures. A curved needle, armed with a strong double ligature, was passed through the organ in the median line, a little distance within the mouth, and then each ligature was tied as far back as possible, so as to leave a kind of tip or projection to the portion of tongue remaining in the mouth. The little patient did not appear to suffer that acute pain which was expected to have been caused by this operation; she ceased to cry as soon as the

ligatures were tightened. She passed a quiet night, and was cheerful and lively on the following day; the protruding portion of the tongue was cold and purple; the part within the mouth was not at all inflamed or sore when touched.

April 21.—The protruding portion has become warmer, probably from a partial return of the circulation, permitted by a loosening of the ligatures caused by diminution in the bulk of the organ, consequent upon the discharge of a quantity of serum; for, on slightly abrading the surface, blood oozed from the part. In consequence of this, Mr. Hodgson applied fresh ligatures, in the same manner and in the same situations as on the preceding day. This stopped the bleeding, and the part again became cold. There does not appear to be any untoward symptom. The mouth is frequently washed out, by means of a syringe, with yeast and water, to which a little tincture of myrrh is occasionally added.

22.—The part is still cold and black. There are no signs of inflammation in the mouth; the child seldom cries, and seems to be going on as well as possible. Ten, p.m.—Some degree of warmth has returned in the part anterior to the ligatures, and on rubbing the surface a slight bleeding took place from the sloughing part. The ligatures were accordingly renewed as before. During this proceeding the little patient did not appear to suffer any pain.

23.—The protruding portion of the tongue is black and cold; the circulation is altogether stopped in it; the slough has nearly separated; no inflammation in the mouth; no constitutional disturbance of any importance.

24.—The slough was removed this morning by cutting it away with the scissors anterior to the ligatures, which were quite loose and unattached, so that they came away immediately afterwards. The part that remains, although quite within the lips, is yet very thick. The horizontal position of the anterior part of the lower jaw and of the front teeth is now also very remarkable. The upper and lower jaws cannot, in front, be brought into contact, partly from this peculiar position and elongation of the

lower jaw, and partly from the thickness of the end of the tongue, which projects upwards in the mouth. A little honey and borax was applied to the ulcerated surface from which the slough was detached.

April 26.—General health continues good. The tongue has much decreased in size, and the child can nearly close her lips.

28.—The child's health continuing good, and the mother being anxious to take her home, she left the hospital to-day. She can now close her lips. The ulcerated surface is nearly healed, and the tongue continues to decrease in size.

In June, 1835, two years after the operation, she was again seen. She was then much grown, and could talk and eat without the least inconvenience. Her health was good and her complexion ruddy. The tongue had undergone no enlargement or morbid change since the operation. The pointed tip or end was thick, and stood up in the mouth, but it caused neither pain nor inconvenience. The lower jaw still projected, and the front teeth of the two jaws could not be placed in apposition.

CASE OF

HYPERTROPHY OF THE TONGUE.

BY

T. P. TEALE, Esq., F.R.C.S.E.,

SURGEON TO THE LEEDS INFIRMARY.

COMMUNICATED BY

JOSEPH HODGSON, Esq., F.R.S.

Received June 14th.—Read June 14th, 1853.

In 1848, Emma Lockwood, æt. 1 year, after an attack of chicken-pox, suffered from a thrushy state of the tongue, which became swollen, and protruded slightly beyond the teeth for two or three weeks. After an interval of two months, the tongue again became swollen, and continued to enlarge slowly until the child was two years old. At this time she was admitted into the Leeds Infirmary under my care. The tongue then projected beyond the teeth, forming a tumour the size of a walnut, and deeply ulcerated at its under surface from the pressure of the teeth. The lower teeth were extracted; numerous punctures were made into the substance of the projecting swelling, and leeches were occasionally applied, but without any material benefit. The tongue continued to enlarge until the child reached the age of three years, when the protrusion was as large as a pullet's egg, strangulated by the jaws, and perfectly irreducible. Treatment by pressure was now adopted. The protruded part was bound, rather firmly, by circular folds of a narrow bandage of calico. After this treatment had been adopted for about a month, the part was so far reduced in size as to admit of being fairly pushed within the mouth, in

which situation it was retained by a bandage passing over the mouth and tied at the back of the head. This bandage was worn night and day for several months, being only removed when food was taken. She afterwards became impatient of the treatment, and the bandage was occasionally neglected for a few weeks, when the tongue invariably began to protrude and enlarge. The tendency to enlargement has of late gradually diminished, and a very moderate degree of pressure is now sufficient to prevent protrusion. She is six years old, and is in good general health. The tongue is a little harder and larger than natural.

CASE OF

POPLITEAL ANEURISM,

CURED BY COMPRESSION OF THE FEMORAL ARTERY IN THE UPPER THIRD OF ITS COURSE, THE PATIENT DYING FIVE MONTHS AFTER OF ANEURISM OF THE ABDOMINAL AORTA, WITH AN ACCOUNT OF THE DISSECTION OF THE AFFECTED LIMB.

BY

J. MONRO, M.D.,

BATALLION SURGEON, COLDSTREAM GUARDS.

Received Jan. 7th.—Read Jan. 11th, 1853.

THE following case, being the second in which the cure of a popliteal aneurism by compression of the femoral artery, has been accomplished in the Hospital of the Coldstream Guards, is interesting, not only from the circumstance of its adding another to the already numerous list of instances in which this plan has been successful, but from the valuable practical fact which seems to be proved, viz., that a much smaller amount of pressure on the artery communicating with the sac than was formerly judged necessary, will suffice for the cure of the disease. This therefore being established, the chief difficulty which prevents pressure from being steadily applied in practice, arising from the severe pain it generally causes, is materially lessened; the chances of sloughing of the integuments are diminished, and a plan of cure is adopted which is more consonant to the natural process; and consequently a safer one. I am well aware that it will be objected to the practice of not entirely impeding the flow of blood into the aneurism, that it is tedious, and that cases are on record where the sac has ceased to pulsate in three days, and even less when the

tourniquet has been removed, after having been applied for that time, so as to entirely stop the current of blood through the artery; but, in my opinion, we cannot too closely imitate the process of nature, which has been so well illustrated by Mr. Hodgson and others; whereby the abrupt change which must follow the cessation of the circulation through the usual channel, from the undeveloped state of the anastomosing vessels, is avoided, a circumstance the more to be borne in mind, considering the tendency to disease, if not the actually diseased state, of other portions of the arterial system in those in whom one aneurism already exists.

It is with these views that I have now the honour of submitting the accompanying Case to the consideration of the Society:

CASE.—Drummer W. Keynes, æt. 23, of a spare and delicate habit of body, whose health had been evidently impaired, walked to the hospital on the 19th April, 1852, complaining of severe pain in the calf of the left leg, which was also a good deal swelled; the veins were dilated. He stated that five days ago, prior to which he had the perfect use of his limb, he had been playing cricket, when he was obliged to desist, owing to the pain he suffered in the situation referred to. Had not remarked any pulsating tumour, but imagined he had met with a sprain. Thinking that it would get better, he had not applied for advice, and had continued his duty since. Finding that the leg became more painful, he now came to the hospital.

The nature of the disease was at once evident on examining the popliteal space.

A large pulsating tumour, about the size of a small orange, filled that situation. It had all the diagnostic signs of aneurism, more particularly the bellows-sound heard on the application of the stethoscope. The sac admitted of being partially emptied by pressure, but returned to its former dimensions when that was removed. The colour of the integuments was natural. The femoral

artery was examined, with the view of ascertaining whether any other aneurism existed. The artery pulsated soundly from Poupart's ligament to the spot where it enters the tendinous canal of the adductor magnus. The chest was also examined, and found in a healthy condition. The sounds of the heart indicated no disease, nor was there any morbid symptom along the course of the abdominal aorta.

On referring to previous admissions, it was found that he had been many times in hospital, not, however, for any lengthened period, but for slight catarrhs to which he was subject. He had been treated for venereal sores five times; once only had mercury been administered, and then merely for ten days.

The successful issue of a former case, the particulars of which are published in the 'Transactions' of this Society, and which was treated by my colleagues, Messrs. Greatrex and Robinson, encouraged me to make a similar attempt for the cure of the disease, particularly as circumstances seemed to favour the plan. The thigh not being muscular, the artery was easily compressed. With the concurrence of my colleagues and Mr. Holt, who was present, I applied an instrument, invented, I believe, by Mr. Phillips. It was fixed to the pelvis and thighs, and more calculated to steady the pressure which is made by screw pads, constructed to shift up and down the thigh, by an ingenious mechanism, as may be desired. The pressure was at first made immediately below Poupart's ligament, and then only moderately. This was chosen as an eligible situation, especially as there were no enlarged glands. In about an hour and a half the patient complained of much pain, and towards evening became so feverish that it was necesary to remove the apparatus. Being persuaded that this was owing more to alarm than to the pressure employed, which was only moderate, and in order to reconcile him to it, I began next day to make manual pressure a little below the origin of the profunda. This was easily effected by the assistance of several intelligent convalescents, who were shown the

manner of doing it, and who relieved one another in this duty. The artery was compressed to such a degree only as to allow the sac to pulsate feebly. For the first three days this plan was followed for eight hours during the day only. The patient did not complain of the pain, nor did it seem to produce any feverishness. This being the case, he said he thought he could bear the pressure with the clamp tourniquet. This was, therefore, placed on the thigh, the same principles being attended to in its use. It was removed at night under the impression that it would almost certainly be displaced before the patient became accustomed to it. Finding that it was borne more easily than was expected, the instrument was kept on during the night also from the 26th.

The pressure was not always made in the same place. When there appeared to be any tendency to excoriation of the skin, the pad was shifted either higher up or lower down as the case might be. The report of the 30th states that no pain or feverishness had been occasioned by the instrument; the sac had become harder, and sensibly diminished in size.

The œdema of the calf was less; and he complained no more of pain in that situation. The temperature of the affected limb was like the other. Two of the articular arteries were perceived pulsating over the inner condyle of the femur and head of the tibia. The pulse was 70. Neither the dorsal artery of the foot nor the posterior tibial could be felt. On the 3d of May all had gone on well since last report, the patient bearing the pressure of the tourniquet without suffering. The pulsation of the aneurism had become feebler; the instrument having been removed for a few minutes to ascertain this. The œdema of the calf had diminished, and there was no pain in that situation.

On the 6th, the sac ceased to pulsate, although no greater degree of pressure was used. With the view of finding whether this state would continue, the tourniquet was taken off. The sac did not pulsate for an hour

afterwards; but, at the expiration of that time, it began to do so feebly. The instrument was, therefore, re-applied, and pressure made as before. The sac had continued to shrink since last report, and become more solid.

The measurement around the joint had diminished from $16\frac{1}{4}$ to $14\frac{1}{2}$ inches. On the 8th, the pulsation in the sac did not return when the tourniquet was removed. It had ceased entirely. The instrument was, therefore, laid aside.

All that was considered necessary to be done now was to keep the patient from using the limb for a short time. He went on favorably from this date: the absorption of the contents of the aneurism proceeded; the functions of the limb were restored; its appearance did not differ from the other; and the anastomosing vessels had become more developed. The femoral artery was ascertained to be pervious to the tendinous canal; but the pulsation of the dorsal artery of the foot and posterior tibial could not be felt. He was not allowed to leave his bed till the 2d of June, when the sac had contracted so much that there seemed to be little risk of allowing him to walk about. He gradually acquired the perfect use of the limb, and was dismissed to his duty on the 2d of July, some thickening only being felt around the remains of the popliteal artery; but the pulsation of the posterior tibial and dorsal artery of the foot could not be perceived. He continued to do his duty well till the 9th of November, when a small abscess formed over the patella of the leg which had been affected. He remained a few days in hospital with this, and was dismissed.

On the 19th of the same month, he was again admitted with severe pain in the loins and abdomen, a sense of coldness and numbness of lower extremities, small and quick pulse, and the general aspect sunk and haggard. On inquiry, it was found that he had given way to intemperate habits on leaving the hospital. The cause of the symptoms was immediately discovered to be an aneurism of the abdominal aorta about the situation of the cœliac artery. It

had formed subsequently to his dismissal from hospital in July. The tumour appeared to be a little larger than the closed fist, and of an irregular oblong shape. The stethoscope left no doubt as to its nature. Such being the case, only palliative remedies were given, with the view of allaying his sufferings. Opiates and the mildest diet were tried. He lingered in great suffering till the 10th December, when he died rather suddenly from the rupture of the sac—the day before the patient having felt extremely faint and low. The *post-mortem* examination confirmed the diagnosis. The heart was sound in every respect, also the aorta, which was free from disease except in the short space between the cœliac axis and superior mesenteric arteries, where a large sac of the shape mentioned above was found. The walls of the sac were very thin, apparently chiefly condensed cellular substance. The openings into it were two, of which one about the size of a shilling had probably been a rupture of the coats of the artery, the edges being partially rounded and partially ragged. The sac was filled with a dense coagulum; several pounds of blood were found extravasated in the cavity of the abdomen. A wax and tallow injection was thrown into the common iliac artery of the limb which had been affected with aneurism, and the following facts were ascertained:

The femoral and popliteal arteries were of the natural size, and pervious until opposite the centre of the popliteal space. The profunda and its branches were much enlarged. Slight thickening was all that remained of the sac, beneath which the artery was obliterated down to its division into the anterior and posterior tibial branches. The anterior tibial was enlarged to the size of the posterior tibial —the latter of the usual size—the peroneal larger than usual. The muscular branches given off by the popliteal to the semi-membranosus, vastus externus, and biceps, larger than generally found. The ramus anastomoticus magnus three times its ordinary size, and dividing into three considerable branches soon after leaving the trunk, any one of which was rather larger than the normal trunk; the inner-

most passing through the vastus internus to the patella; the second, in front of the tendon of the adductor magnus; the third, behind it, anastomosing in the neighbourhood of the patella with the inferior articular and recurrent tibial arteries, and with each other. Two superior articular and azygos arteries given off by a common branch just previous to the remains of the sac; these anastomosing freely with the recurrent tibial, and ascending branches from the posterior tibial and peroneal. The sural arteries given off at the point where the artery is obliterated, impervious for half an inch, the rest of their course having been injected in a retrograde direction. A tortuous plexus in the substance of the popliteal and peroneal nerves supplied from the branch to the semi-membranosus, and uniting below with a recurrent branch from the anterior and posterior tibial behind the head of the fibula. Another branch descending from the azygos articular communicated with recurrent branches behind the head of the bone. Besides this, a branch from the azygos passed down between the popliteus and bone, and inosculated with a recurrent branch from the anterior tibial just before that vessel passes through the interosseous membrane. The inferior articular arteries enlarged, the inner inosculating freely with the anastomotica magna; the external, with the recurrent tibial and superior articular of the same side.

The recurrent tibial was the most enlarged of any of the branches; it was equal in size to the radial artery, and anastomosed freely with both articulars and the plexus in the peroneal nerve. The communication between the external and internal articular effected by branches three times their usual size,—all the communicating branches, especially that in the sciatic nerve, more tortuous than usual. The popliteal vein was obliterated for the space of three inches, probably from the pressure of the sac.

The femoral vein pervious, and nothing of note observed as to its state. The sural veins, from the heads of the gastrocnemius (which lie close on the inner side of the condyles of the femur), were unobstructed. Nothing particular

remarked in the external and internal saphena. With the exception of there being some adhesion of the pleuræ on the left side of the chest, and a little serum in the pericardium and in the peritoneal cavity, the morbid appearances did not show anything remarkable.

A FURTHER ACCOUNT

OF

FATTY DEGENERATION OF THE PLACENTA,

AND THE

INFLUENCE OF THIS DISEASE IN PRODUCING

DEATH OF THE FŒTUS, HÆMORRHAGE, AND ABORTION.

BY

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COMMUNICATED BY

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SINCE the publication of my first paper and appendix in the 34th volume of the 'Medico-Chirurgical Transactions,' I have had the opportunity of examining several additional examples of fatty degeneration of the placenta. Some of these cases exhibit forms of this affection sufficiently distinct from the form described in that paper to merit a separate notice. Several present a form which corresponds closely with the description of Dr. Franz Kilian's case, ('Neue Zeitschrift für Geburtskunde,' vol. xxvii.) One case shows the co-existence of fatty degeneration, with hydatiginous degeneration of the chorion. Two are examples of fatty degeneration of the chorion at a very early period, associated with abortion.

The histories of the cases now submitted to the Society, viewed individually and collectively, will, I believe, be found to throw much additional light upon the pathology of fatty degeneration in an organ in which the existence of that affection has only recently been established. They lend confirmation also to the opinion I have before expressed, as to the importance of this affection as a cause of death of the fœtus, hæmorrhage, and abortion; and they illustrate the modes by which these events are brought about.

I am again indebted to my friend, Dr. Hassall, for his valuable aid in the microscopical investigations, and in the preparation of the pictorial delineations of the diseased structures. I have also to thank many gentlemen, to some of whom I was personally unknown, for having placed many interesting specimens of diseased ova at my disposal.

In numbering the cases I have thought it useful, having regard to future reference, to number them consecutively from those which form the subject of my first paper. These were three in number; the first case in this communication will therefore be numbered as the fourth.

Case iv.—On the 26th April, 1852, Mr. Humby, to whom I am indebted for the two placentas which first led me to the observation of fatty disease of the placenta, left me a placenta with the fœtus attached. The fœtus had apparently attained the middle period of gestation. It was shrunken, as if it had undergone maceration. The cuticle did not peel off; it was of a brownish colour. The cord had a similar appearance. The fœtus and cord were quite free from putrefaction. The lady who was the subject of this case had not been conscious of the death of the fœtus. She was taken suddenly with slight pains. Mr. Humby arrived in time to observe the passage of the child and placenta, which all came together. There had been some offensive discharge before labour; but no hæmorrhage occurred either preceding, during, or after the labour.

The *placenta,* like the child, was free from putrefaction. The maternal surface was deeply divided by sulci. Its general

aspect much resembled that of the brain, both in colour and subdivision. All the lobes had a similar appearance. Pale, yellow, glistening like fat on the prominent portions; pink or red in the sulci. In the centre of some five or six of the lobes was a small dark clot, marking the recent rupture of a vessel connecting the uterus and the placenta. In the sulci between the lobes there was evidence of somewhat more extensive vascular connection with the uterus. With these exceptions, the placenta had generally an exsanguine appearance. A section showed that the yellow fatty appearance was most marked on the decidual surface; on approaching the fœtal surface there was more appearance of blood. There was nothing like fresh healthy placental tissue. The consistence was very firm; it rather broke than tore, and cut like a solid body.

The *villi* taken from any part near the decidual surface were much altered; villi from nearer the fœtal surface were less altered, but none were found perfectly healthy. The villi in the more consolidated lobes showed the greatest amount of alteration: they were brittle, misshapen; the vessels breaking up, and losing their defined outline and form; the chorion was for the most part destroyed, but the nuclei in the walls of the vessels in many places were not, as is usually the case, destroyed, but enlarged and crammed with granules. In the less consolidated lobes of this placenta, the vessels retained their proper size and shape, but the chorion was destroyed to a great extent; the nuclei of the walls of the vessels had disappeared; numerous oil-granules were scattered over the walls, and in addition there were numerous masses of fatty matter, consisting of aggregations of globules, disposed usually in the course of the vessels, but sometimes confined to the extremities.

The following appears to me to be a rational exposition of the pathology of this case. The fœtus evidently perished some time before its expulsion. All fœtal-placental circulation had therefore ceased. The large vessels which still connected the placenta with the uterus were the channels by which an imperfect utero-placental circulation was carried on

up to the last. As there could have been no interchange between maternal and fœtal blood, this circulation must have been simply to maintain a low degree of nutrition in the placenta. It is reasonable to infer that the granular degeneration had commenced prior to the death of the fœtus; that its gradual advance having rendered the villi unfit for the interchange of the elements of the maternal and fœtal blood, had thus destroyed the fœtus. A certain connection between the uterus and placenta was still kept up. The granular conversion of the villi continued to increase after the death of the fœtus, until it had reached such an extent as to cut off so much of the remaining vascular communications as to leave the longer attachment of the placenta to the uterus impossible.

This gradual arrest of the circulation in the placenta, and the gradual obliteration of the utero-placental vessels before the detachment of the placenta, will explain the fact, that there was no hæmorrhage at the time of labour.

Case v.—Mrs. R—, a patient of Mr. Barker, was taken in labour on the 25th of October, 1851. Considerable hæmorrhage in gushes occurred at the commencement. Mr. B. found the placenta at the os uteri partially detached. The funis was prolapsed. He aided the expulsion of the fœtus. The placenta was partially adherent, so that it was extracted with some difficulty. The uterus contracted well; and the patient recovered favorably.

The *fœtus* was near the full size. Its abdomen was distended with fluid; the skin dark coloured; the cuticle easily detached. Some time before labour the patient had ceased to feel the movements of the child: she was only conscious that it "fell about" in the womb. She knew that it was dead. She had had one living child some years ago; but several succeeding pregnancies have terminated in the same manner as the one now described. In one labour it was said that the placenta was morbidly adherent, and had to be detached by the hand, much hæmorrhage attending.

The *placenta* in this instance was free from decomposition. Its bulk was considerable; it had been a good deal torn in extraction. The greater part presented to the naked eye a healthy appearance. In parts, however, there were whitish glistening masses, interspersed in the ordinary red structure. In some parts, also, the tissue was soft, and easily lacerable. In two different parts there was a mass of firm coagulated blood, evidently of a date anterior to the labour. The cord was dark-coloured, flaccid, and distended with turbid serum.

The microscopical examination of the white portions revealed the same form of fatty degeneration as that described in my former paper—viz., the villi were mostly exsanguine, opaque, thickly studded with spherules of oil; the chorion was thickened, and destitute of nuclei. Similar appearances were also observed, although to a less marked degree, in portions of the placenta which, to the naked eye, appeared to be healthy.

There were two complications in this case, which render it difficult to determine what share the fatty degeneration of the placenta had in producing the death of the fœtus and the hæmorrhage. The placenta was partially attached to the os uteri, and a part morbidly adherent to the walls of the uterus. This morbid adhesion and the soft, lacerable texture might indicate some previous inflammatory action; and there may appear to be some ground for supposing that the fatty degeneration was the consequence of this inflammatory action. The partial attachment of the placenta to the os uteri was probably the cause of the two masses of firm coagulated blood, the result of a partial separation some time prior to the labour. The further separation of the placenta from the os was almost certainly the cause of the hæmorrhage at the commencement of labour. The condition of the child and the state of the cord clearly show that all fœtal circulation had ceased for some time prior to labour. The condition of the placenta observed after expulsion and the hæmorrhage show that considerable vascular connection had been kept up between

parts of the placenta and the uterus. The effusion of fluid in the peritonæum of the fœtus might lead to the conjecture that disease of the fœtus had been the cause of its death.

In the presence of so many complications, I am anxious not to draw any forced conclusions, and not to attribute the hæmorrhage and the death of the fœtus to any one pathological condition to the exclusion of the rest. But, upon taking into consideration the loss of several fœtuses in succession, and how much this circumstance is in accordance with the history of most of the cases of fatty degeneration of the placenta I have met with, I am not disposed to reject altogether the opinion that a similar cause may have been in operation in this instance.

Case vi.—On the 10th of June, 1852, Mr. Humby brought me another placenta. A young lady, in excellent health, was delivered, after a very easy labour, of her first child. There was no hæmorrhage. The child was putrid, the cuticle easily detaching. It had reached the size of maturity. She had observed no symptom indicating the death of the child.

The *placenta* was very small; the weight $7\frac{1}{2}$ ounces. It was divided by deep sulci into three principal lobes, and three or four smaller ones. One large lobe and a smaller one were pale, and showed no mark of recent connection with the uterus. Another of the larger lobes had more of the aspect of normal placenta. It had a pale reddish colour, a pulply feel, and there were several fresh venous coagula hanging out of the semi-circular openings on the decidual surface. The rest of the placenta was of a character intermediate between those of the portions above described. The substance of every part was tender, softer than natural, lacerable. It was generally pale and greasy-looking. There was no consolidation in any part.

The membranes were green from commencing putrefaction. The cord was flaccid, and infiltrated with reddish

serum. There was no change of a putrefactive kind in the substance of the placenta.

Under the microscope, the villi were observed to separate readily; but none were discovered perfectly healthy. In some the nuclei of the chorion and of the vessels were perfect in form, and contained only a few granules. The extremities of many villi were enlarged, and contained much fatty matter in the shape of minute molecules and large spherules or droplets (see fig. 1), giving the extremities, when viewed with a low power, a dark nodulated appearance. The chorion and coats of the vessels were generally studded with oil-granules. The general colour was dull yellow. No blood was discovered in the villi with the altered extremities. In many of the others some blood was seen.[1]

The above changes were observed in every part of the placenta; but they were most advanced in that lobe which showed no mark of recent vascular connection with the uterus. Generally the change of structure was most marked near the maternal surface.

I infer, from the examination of the placenta, that in this case the progress of the degeneration of the placenta had cut off the embryo. The presence of blood in many of the villi which were less altered in structure indicated that there had been a fœtal circulation at no remote period; and the evident vascular connection of the maternal placenta with the uterus leads to the same conclusion. The reason why there was no hæmorrhage may be more conveniently discussed hereafter.

Case vii.—On the 1st of September, 1851, my colleague, Mr. Forbes, sent me a placenta for inspection, with the

[1] This condition resembles very closely that described by Dr. Franz Kilian. In the majority of instances, the fatty matter assumes the form of granules; but in some cases, it is observed in both forms. In no instance, however, have we observed fatty matter in the form of droplets *alone*. Wherever the droplets have been noticed in the villi, the coats of the villi have been affected by the granular form of degeneration

following statement:—A lady was delivered of a dead fœtus at about five months from the beginning of gestation. She had had hæmorrhage almost from the commencement. This was her third miscarriage. The *fœtus* had attained the development proper to the fifth month of gestation.

The *placenta* was fresh. It had not been immersed in any fluid. The fœtal surface, as seen through the membranes, generally presented a glistening pale-yellow appearance, resembling fat. The maternal surface was deeply divided by sulci, as is mostly observed in the immature placenta. More than half had a pale-yellow glistening fatty appearance. This was more marked in the superficial portion than in the deeper portions exposed by section. It cut and tore like placental tissue, but differed in being comparatively pale and bloodless. The above description applies to by far the larger portion of the placenta. But there was a *second* portion, consisting of one lobe, quite distinct from the rest, of a darker colour, and which cut like flesh. A section of this showed engorgement of blood, extending to extravasation. There was also a *third* portion, which differed from the rest. Along the margin for nearly half the circumference the colour was darker and the consistence firmer than that of the general mass. There was the appearance of recent effusion of blood and fibrine.

Of the *first,* or larger portion of the placental mass, one lobe, which was white and almost perfectly exsanguine, exhibited, under the microscope, universal fatty degeneration in the extremities of the villi, which were dilated and misshapen from the presence of the deposit. Two other lobes of this portion, which exhibited the like alteration in many of the villi, but to a less extent, were of a light pink colour. The villi contained a small quantity of blood.

In the *second* portion, consisting of a distinct lobe, the villi were much distended with blood; some were varicose or irregularly dilated. There was also the same granular alteration as in the first portion, but to a less extent. This portion was intensely congested.

The *third*, or marginal portion, appeared consolidated or hepatised. Under the microscope, fragments of villi were seen, in some of which the same granular alteration, as in the *first* portion, was observed. But the chief part of the substance was composed of effused fibrine, itself degenerating into granular matter.

I have little doubt that this case was one of primary granular degeneration of the villi, leading to the death of the fœtus and abortion. The villi in every part were affected. The engorgement with blood and the varicose appearance of the villi of the second portion would seem to warrant the inference that an unusual effort had been made in this lobe to compensate for the almost complete cessation of circulation in the rest of the placenta. The hepatised appearance and the effusion of fibrine in the third, or marginal, portion were probably the result of a similar excess of action at an earlier period. Possibly they might have been the result of the giving way of the diseased coats of the villi under the additional strain imposed upon them by the imperfect functional capacity of the main portion of the organ. In the main portion there was no evidence of the presence of anything but the ordinary tissues of the placenta, in a state of more or less advanced granular degeneration.

Case viii.—On the 8th of October, 1852, Mr. R. R. Robinson, of Camberwell, forwarded me a portion of placenta, and subsequently communicated the following history. Mrs. —, aged 31, a delicate lady, had been the subject of disease of the spine some years ago, which was cured without deformity. She was subject to a short hacking cough; but no disease of the lungs could be detected. She had been married four years. Eleven months after marriage she was delivered of a still-born child after a seven-months' pregnancy. Fourteen months after her first confinement she was again delivered of a still-born child after a gestation of six months and a half. She was very weak for a long time afterwards. For the last two years she had

always a red tongue, and was subject to diarrhœa. In May, 1851, she had an attack of abdominal inflammation, from which she recovered so far as to feel better than for a long time previously. In February last she became pregnant again, and went on very well till the 8th of June, when, having gone out in a carriage, she was much shaken; suddenly some hæmorrhage occurred, attended with uterine pain. In the night this returned. For some weeks she kept in bed, and then was removed to the sofa. Under mild nourishment and the use of steel she derived considerable benefit. On the 1st of October Mr. Robinson was requested to see her. She complained of feeling very faint; and not having felt the child for a day or two, she was fearful it was dead. By the use of ammonia, the faintness went off, and she thought she felt the child. On the morning of the 6th, however, she complained of violent pain in the back and sides, and decided labour-pains came on, but subsided. They returned on the following day; a gush of blood came, and a continued draining ensued. The membranes were immediately ruptured, when a large quantity of liquor amnii, not fetid, escaped. The pains increased; there was no further hæmorrhage; and the child was born in twenty minutes. It was a male of seven months' growth; it had evidently been dead some time, as the cuticle peeled off. The placenta quickly followed; the uterus contracted well. Some clots followed: the blood was dark. The cord was thick, red, and gelatinous. The urine was unaffected by heat or nitric acid.

The *placenta* was rather small, easily torn, and soft. The torn surface was very pale, moist, and shreddy. The portion sent to me by Mr. Robinson was of a uniform dull opaque yellow fatty appearance. It presented on two or three points of the uterine surface marks of recently severed vessels, indicating a limited connection with the uterus. The texture was not at all consolidated; it was generally bloodless. Under the microscope, the villi were seen to be mostly exsanguine. The nuclei along the margin of the chorion were visible, but dotted with granules. The chorion

was not detached from the vessels, but was generally affected with granular degeneration. Numerous extremities of villi exhibited the black opaque granular aggregations observed in Case VI, and represented in fig. 1. As in that case, the villi showing the nodular aggregations contained no blood.

The appearance of the child and the placenta, and the history of the case, place it beyond a doubt that the child had been dead at least ten days before birth. The presence of blood in the villi least affected, and the recent connection of the maternal placenta to the uterus, led me to infer that the child had been cut off by the advancing degeneration of the fœtal portion of the placenta. The hæmorrhage which occurred on the 8th of June—that is, in the fifth month of pregnancy—after succussion, was in all probability owing to partial alteration of the placenta, which rendered it readily liable to partial detachment. There is no reason to suppose it to have been owing to abnormal position.

The impaired health of the patient, as bearing upon the state of nutrition, is worthy of remark.

Case IX.—On the 17th of June, 1852, a patient of the Western General Dispensary, aborted at about the tenth week of gestation. She ascribed the abortion to emotion consequent upon her mother's death. She had been treated for dyspepsia previously. There was a good deal of hæmorrhage. The ovum came away easily. She had noticed nothing to lead her to suppose she was going to abort.

The *decidua* and *chorion* were undoubtedly of an earlier date than ten weeks. I conjectured, from their appearance, that development had been arrested at about the seventh week. The decidua had, however, retained some vascular connection with the uterus to the last. No embryo was discovered. The *decidua* was very much thickened, and somewhat infiltrated with recent blood. On the uterine surface the oblique vascular openings were distinct; but it

was pale, and had a fatty appearance. Magnified 420 diam., the cells of the decidua were well marked: most had a caudate elongation at either end. By no amount of laceration could these cells be separated from the fibrous membrane in which they were imbedded. The caudate elongations seemed to interlace with each other, and to effect a firm hold upon the membrane. Numerous granules of oil were seen in the cells. The fœtal surface of the decidua presented similar appearances; but the fatty change was less extensive.

The villi of the chorion, viewed with the naked eye, were of a dull yellowish-white colour. They did not freely float out in water. They presented numerous nodular enlargements at the extremities. With an inch glass these enlargements were seen more clearly: they looked like pear-shaped bodies on very narrow stalks, and greatly resembled minute hydatids of the chorion which have shrunk from being kept a day or two. With a quarter-inch glass these enlargements were found to contain granular fat; they were opaque, some quite dark, not always regular in shape, but mostly pyriform. The villi generally presented the nuclei, if not entire, not quite destroyed. Granular fat was abundant throughout. It was clear that the degeneration of the chorion was more extensive than that of the decidua.

The pathological history of this case may admit of some discussion. Whether there is any relation between the peculiar conformation of the villi and the granular degeneration is a matter of doubt. It must also be difficult to determine whether the deviations of form and the alteration of structure of the chorion preceded or followed the destruction of the embryo. But there is little ground to hesitate in concluding that the degeneration of the chorion preceded that of the decidua. This slow progress of the disorganisation of the decidua — an essentially uterine structure—will account for the continued vascular connection with the uterus, and the retention of the ovum for a period of three weeks or more after the destruction of the embryo.

I think this is the most convenient place to make a few observations upon the singular conformation of the villi in this case. Vrolik has reproduced a plate from Seiler, with the following description:—"Flocci secundùm Seiler à 5â vel 6tâ graviditatis hebdomade, vesiculis terminati, *quæ dein evanescunt*, sed nonnunquam abnormi ratione permanent et tunc isti spuriæ graviditatis speciei ansam dant, quam molam botryoidam vel hydaticam dicunt. Non opus est monere etiam hac periodo nulla adhuc in chorii floccis formata esse vasa." On comparing the figure of Seiler with the appearance of the chorion in this case, a striking resemblance was observed. It was figured by Seiler and is referred to by Vrolik as the normal form of the chorion at an early period. Désormeaux has described a similar appearance, and attributes the origin of hydatids to the persistence of these apparently vesicular enlargements of the chorion. That the chorion figured by Seiler was diseased is quite certain, and I am disposed to believe it had undergone fatty degeneration.[1] The chorion is evidently thickened and partially detached; these are changes characteristic of fatty degeneration. It will be interesting to compare this figure of Seiler's with fig. 2, illustrating the next case.

Case x.—On May 14, 1852, I was called on to see Mrs. H—, a somewhat delicate but healthy lady, who had borne two healthy children at the full period. She had symptoms of abortion at a very early period of gestation. She had been in her usual health on the 10th. For three days she had rather profuse hæmorrhage, which came on without any known cause. She had constant pains; coagula passed at intervals, and there was continual draining. On the second day a portion of chorion, covered with shaggy villi, was passed. On the third day another portion of chorion, with villi covered with minute hydatidiform cysts. Shortly afterwards a portion of decidua, slightly ecchymosed, but otherwise seemingly healthy, came away. No trace of embryo was discovered.

[1] See Seiler's Plate, fig. 2.

A little hæmorrhage still continued. Shortly afterwards, as she was being lifted out of bed, more hæmorrhage occurred, and a mass as large as a small orange came away. This mass consisted of decidua infiltrated with recent coagulated blood, and chorion with numerous cysts. Still there was no embryo. After this the hæmorrhage ceased, and no further portions of membranes were observed. The patient went on favorably until the 18th, when, having sat at an open window, she had a stiff neck, acute pains in the left groin, and stiffness in the left leg. She was unable to move the left leg without increasing the pain in the groin and left pelvic region. There was considerable febrile movement and constipation. Leeches relieved the local symptoms; a dose of calomel brought away a large quantity of fæces. On the 22d she was much distressed by cough. Auscultation revealed no affection of the chest whatever. I have witnessed in other cases of abortion a most distressing irritative cough supervene, when there had been no symptom of the kind previously, and when every care had been taken to avoid catching cold. It appears to me that the cough, in these instances, is to be ascribed to irritation propagated from the uterine mucous membrane, and that it is analogous to the cough excited by irritation of the mucous membranes of other parts. In this particular case, no relief was afforded by the usual remedies, but the application of galvanism to the chest was attended by the most marked beneficial effects. Mrs. H— gradually recovered her strength.

Examination of parts of the ovum.—A portion of the chorion was entirely free from hydatiginous degeneration. When this portion was viewed with a strong lens, the villi looked of a dull, opaque, yellowish-white. They were more rigid than healthy villi. Some villi were pure white. Some of the ends of the villi were thickened, nodular, and misshapen. Under the microscope, the vascular structure of the villi precisely resembled the villi of the mature placenta in their general character. Where the structures had not undergone much change, the nuclei in the chorion along

the margin were distinct; but more or less deposition of minute oil spherules was general throughout the structure of the villi. In some, where the change was more advanced, and where the chorion yet remained covering the vessels, it was considerably thickened, opaque, and of a yellowish tinge; the nuclei had disappeared, or nearly so, and the membrane was studded with oil spherules. In parts, the chorion, rendered brittle by change of structure, had become detached from the vessels, which were seen here and there partly denuded, with portions of broken chorion adhering in the form of portions of tube (see fig. 2). When the vessels were completely bare, their walls were discovered to be also affected by fatty degeneration. The vessels contained no blood.

The concurrence of hydatidinous and fatty degeneration in this case is interesting. The progress of either affection would have sufficed to destroy the embryo and lead to abortion; but, judging from the universality of the fatty degeneration and the generally exsanguine condition of the villi, I am disposed to consider this as the chief cause. The decidua was healthy, and had evidently maintained its connection with the uterus to the last. This circumstance, which was satisfactorily ascertained, serves to fix the cause of the abortion in the chorion. A point worthy of observation, is the very early period (probably not more than six weeks) at which fatty degeneration may be developed in the villi.

In connection with this case I will hazard the suggestion, that fatty degeneration may be the process by which those villi of the chorion, whose function becomes superseded at an early stage by the concentration and development of the placenta upon one portion, become atrophied and disappear.

The cases now observed, I believe, furnish illustrations of the influence of fatty degeneration in producing abortion, by rendering the villi of the chorion or placenta unfitted for their office of maintaining the nutrition of the embryo. They also illustrate the relation of this affection to hæmor-

rhage. I would especially point to Case x as an example of abortion brought about by this disease proceeding to an advanced stage in the villi of the chorion at a very early period of gestation, and before the formation of a distinct placenta; and Case ix might probably be adduced as a similar instance. Case vii is an example of the disease leading to the death of the fœtus, and subsequent expulsion of the ovum in the fifth month. Case iv is an example similar to the preceding, the death of the embryo and abortion occurring at about the end of the fifth month. In Case viii, Mr. Robinson's, and in Cases i and ii in my first paper, the death of the embryo seems to have occurred in the seventh month of gestation. The case of Dr. Franz Kilian exhibits death of the fœtus and expulsion at the end of eight months. Cases v and vi afford examples of destruction of the embryo in the last month of gestation. It thus appears that fatty degeneration may cause the destruction of the embryo, and consequent abortion, at any period, from the earliest to the latest, of gestation. That the fatty degeneration of the placenta was really the cause of abortion in most of the foregoing cases, appears to me to be established by the following considerations:

In the first place, on referring to the histories of the cases related, it will be observed that in no case was there any sign of decomposition of the placenta.

Secondly, the distinct indications of continued vascular connection between the placenta and the uterus up to the moment of detachment and expulsion.

Thirdly, the various degrees of progress of the degeneration in different portions of the same placenta, some portions being far gone towards disorganisation, whilst in others the villi remained comparatively healthy—to the naked eye entirely so—although exhibiting, under the microscope, evidence of the same change in an early stage: these portions still showing, by the presence of blood within the vessels, a sure indication that they had recently fulfilled their allotted functions.

Fourthly, the obvious commensurate diminution of the

vascular connection between placenta and uterus with the progress of the affection, especially exemplified in Case IV.

Fifthly, the almost constant evidence of the death of the fœtus having taken place at some definite but not remote period, usually about fourteen days, before expulsion; and the improbability of this affection of the placentas having commenced and attained the very advanced condition observed within that limited period.

Sixthly, there is the analogical argument derived from the better known pathology of fatty degeneration in other organs, as in the muscular structure of the vessels, heart, in the liver, in the kidneys, and in the blood-vessels. The circumstances under which this destructive change is observed in these instances preclude all doubt that it arises during the life of the individual, and that sooner or later the resulting disorganisation terminates in death. Is it reasonable to conclude that the placenta is the only organ in which identical appearances of structure can be produced as a *post-mortem* change?

That a particular form of disorganisation, or rather metamorphosis of a fatty character *may* take place after the death of the placenta, is beyond a doubt. I have myself recorded a case illustrating the fact in the Appendix to my first paper. But the appearances exhibited in this undoubted example of post-mortem granular metamorphosis, are essentially distinct from those observed in all the other cases, and represented in the figures accompanying this and the former paper. One character alone will serve to contrast the fatty degeneration of the living placenta with the granular metamorphosis of the dead organ. In the latter, the change of structure is universal and uniform, affecting alike the chorion and the decidua, the whole extent of the vessels as well as their extremities. To my mind the widely-different characters of the two forms of granular change observed, furnish the most absolute of all the proofs in support of the conclusion, that in the cases now described the structural changes took place during the life of the placenta.

In relation to the question of the influence of fatty

degeneration of the placenta in bringing about abortion, I think it of great interest to analyse the steps by which the detachment and expulsion of the embryo and secundines are usually effected in those cases in which the embryo perishes prematurely in the uterus. I have referred to the fact that symptoms indicating the death of the fœtus mostly precede its expulsion by some definite period. An examination of the histories of many cases of premature labour, in which the child was born dead, recorded by Mauriceau, Lee, Crosse, and others, fully bears out this proposition. When the fœtus perishes, the first effect is necessarily the arrest of the fœtal circulation. The fœtal blood no longer flowing through the umbilical capillaries of the placenta, the stimulus to the afflux of blood to the maternal portion of the placenta is lost, and the utero-placental circulation soon ceases also. This is the first step. The next step consists in the gradual atrophy of the placenta, of the utero-placental vessels, and the decidua, which still tend to preserve the adhesion of the ovum to the uterus. Why a certain connection is kept up between the placenta and the uterus some time after the placenta as an organ subservient to the nutrition of the embryo has become useless, may, I believe, be accounted for as follows: The fœtal portion of the placenta perishes with the embryo; but the maternal portion, the decidua, is essentially an uterine structure, and tends for some time longer to preserve its relation to the uterus. This is especially remarkable in cases of early abortion, where the cause appears to have resided in the chorion or the embryo. At the very early period of gestation the connection of the decidua with the uterus is of the most intimate kind, and it is altogether distinct from the chorion. In such a case, a dead ovum may be retained in the uterus for a longer time than in the case of death of the fœtus at a more advanced period of gestation. Case IX may be taken as an illustration of this: the separation of the decidua was probably not effected in less than three weeks.

Whilst the bonds of attachment between the placenta and the uterus are becoming thus gradually impaired, and the

detachment of the placenta is being prepared, important changes are going on in the uterus to prepare for the *expulsion* of the blighted ovum. During the growth of the ovum the developmental stimulus attracts blood in abundance to the uterus. But when the fœtus has perished, and in the course of retrogressive cessation of action the placenta has next become a dead mass, blood is no longer attracted to the uterus. When growth ceases, atrophy begins. In all probability that change which Mr. Rainey has so well described as taking place in the uterus after normal parturition, begins as soon as the contained embryo dies. The muscular element undergoing fatty degeneration gradually recedes. The uterus tends slowly to contract upon itself; its capacity to diminish. So soon as the walls of the uterus have so far collapsed as to press upon the contained ovum, the diastaltic function is excited, and active contractions —labour-pains—complete the detachment, and effect expulsion.

There is an interesting confirmation of this theory. In the case of twin-pregnancy it occasionally happens that one fœtus perishes at an early period of gestation, whilst the other lives on, and the whole may be retained till the full term. In these cases the dead ovum is not cast off, because the developmental activity of the uterus is kept up by the living embryo. Cruveilhier has related an interesting example of this nature, and he has figured the two placentas connected with the dead and the living fœtuses. The one connected with the dead is represented as yellowish in colour, of a tubercular appearance, very compact and atrophied, and it was evident that for a long time all adhesion had been destroyed. I refer to this description for the double purpose of remarking, that, notwithstanding the atrophy of the placenta, and the absence of all adhesion, it was still retained; and also of suggesting the extreme probability, that the condition of the placenta described and figured by Cruveilhier was in reality that of *post-mortem* granular metamorphosis resembling the case described in the Appendix to my paper in the 34th volume of the 'Transactions.'

I will now consider the relation of fatty degeneration of the placenta to hæmorrhage. On referring to the histories of the cases related, it will be observed that in some, hæmorrhage occurred prior to the accession of labour; and that, in others, there was a singular immunity from hæmorrhage throughout. In cases I, IV, VII, IX, X, hæmorrhage took place at different periods of gestation, or on the accession of labour. In cases II, V, and VIII, no hæmorrhage was observed.

I think I am justified by further experience in adhering to the explanation of the mode in which hæmorrhage is caused by fatty degeneration of the placenta given in my first communication. In many cases the affection proceeds with very unequal rapidity in different portions of the placenta. Some lobes may be far gone, while others do not to the naked eye present any obvious departure from the normal structure. In the altered portions two remarkable conditions may be observed. First: they differ from the normal placenta in consistency; they are no longer spongy and yielding, but of more or less solidity. Secondly, the vascular channels between the placenta and uterus are more or less completely obliterated. The consequence of these two conditions is a great disposition to detachment at the diseased points. If the detachment were strictly limited to the diseased points, where little or no vascular connection with the uterus remains, there would be no hæmorrhage. But this can seldom be the case: the detachment of the diseased lobes is almost sure to entail partial detachment of portions still maintaining a freer vascular connection, and thus hæmorrhage will result. This event may be the immediate occasion of the expulsion of the ovum; or, on the other hand, it may be arrested for a time, and recur several times before labour.

In some cases fatty degeneration seems to invade the entire decidual surface of the placenta with great uniformity. In such cases the consistency of the placenta not only remains equal throughout, but may be soft and lacerable; and there is no portion more disposed than the rest to become detached from the uterus. At a certain stage of the

progress of the disease, the embryo will have perished, and all fœtal-placental circulation will have ceased. The great cause of vascular activity in the placenta will be at an end; and any vascular connection between the uterus and placenta still remaining, must be confined to such a sluggish circulation as may suffice to preserve a modified nutrition in the placental tissue. But the placenta is essentially a caducous organ; and when its proper function has terminated, it tends rapidly to break off. The fatty degeneration of the placenta probably goes on at an accelerated ratio after the death of the embryo, the vascular intercourse between placenta and uterus becomes rapidly less and less, until at a period seldom protracted beyond fourteen days, the decidua, the immediate bond of union, is so altered as to have become no longer fitted to preserve cohesion with the uterus, and the vessels which tended to unite the placenta to the uterus, are so far obliterated, that when the placenta drops off, as an etiolated leaf from a plant, not a drop of blood may flow. But although the vascular intercourse between the placenta and the uterus at the period of scission may be reduced to a very inconsiderable extent, I have not witnessed a case in which it was altogether arrested. A few recent coagula may always be seen in the severed utero-placental vessels, although no evidence of blood may be detected in the fœtal portion of the placenta.

It would be foreign to the purpose of this paper to enter into any discussion as to the general pathology of fatty degeneration; but I wish to submit a few remarks specially bearing upon the relation of this disease to the placenta. By far the most interesting question, and at the same time the most obscure, is the *origin* of this affection. It is obvious that, in the improvement of our knowledge of the causes and conditions which lead to the development of fatty degeneration, our hope of discovering rational and successful prophylactic measures depends. It is reasonable to suppose that the primary condition determining fatty degeneration in the placenta may arise in the mother or in the embryo. I will pass over the consideration of the

strictly maternal sources with a simple reference to the marked influence of pregnancy in altering the constitution of the blood—sometimes seriously impairing its quality—and the consequent hazard of defective nutrition both in mother and embryo.

The circumstances which lead to abortion through a primary influence upon the fœtus have been greatly overlooked; while the influence of external circumstances acting upon the mother has been in a corresponding degree exaggerated. In the particular instance of fatty degeneration of the placenta it is also possible to attribute an undue importance to the agency of maternal causes. It seems highly probable that an imperfection in, or loss of, developmental force in the embryo may be the frequent occasion of degeneration of the placenta. It is impossible not to recognise the embryo, from the moment of impregnation, as an independent living organism; and it is not altogether fanciful to represent the new being as finding in the uterus a nidus and the pabulum requisite for its nutrition. If the developmental force originally imparted to the germ be defective,—whether the defect arise from the maternal or the spermatic element,—at a more or less advanced period of gestation, the power of assimilation and nutrition in the embryo may flag; and this failing power of assimilation and nutrition in the embryo may be regarded as the immediate occasion of the degeneration of the placenta. This view is strengthened by the fact, clearly established by the investigations Dr. Hassall and myself have made, that this degenerative process begins in, and causes, the most destructive changes in the villi of the chorion.[1]

Reflecting on the important part which fatty degeneration plays in bringing about the restoration of the uterus after parturition to its normal condition, and upon

[1] The hypothesis here advanced appears to me strictly in accordance with the views of Professor Owen, on the perpetuation of the "spermatic force" throughout different stages of development of the same individual, or of its propagation through a series of different individuals.

other similar phenomena in the history of this affection, I was at an early period of my researches led to conjecture that the placenta might be prepared for being cast off from the uterus by a similar process. The observation that the change is usually, if not always, most marked at the decidual surface, supports this conjecture. I have already hinted that the superfluous villi of the chorion of the early ovum may be removed in this way. In order to ascertain how far this view may apply to the mature placenta, I have examined a considerable number of healthy placentas expelled at the full term. The presence of a certain amount of fat is constantly observed; but the observations hitherto made do not authorise the conclusion that the fall of the placenta is prepared for by a process of fatty degeneration.

The descriptions of the microscopical appearances in the cases related in this and the former paper clearly point to an altered condition of the elementary structures of the placenta similar to that condition of the heart, liver, kidney, and muscles, which is expressed by the term "fatty degeneration." I have used this term in reference to the placenta precisely in the sense which it is understood to convey when applied to other organs. I have thought this explanation necessary in order to distinguish the affection which it has been my object to describe from other changes in the placenta which might be, and indeed have been, a source of confusion. The products of inflammation—effusions of fibrine and coagula—it is well known are prone to undergo a process of fatty conversion. Such are frequently found in the placenta; and I have had numerous specimens of this character brought to me under the belief that they were instances of fatty degeneration of the placenta. Fibrinous deposits, solid, pale-yellow, and, when seen through the fœtal membranes, bearing a close resemblance to masses of fat, are commonly observed on the fœtal surface of the placenta. Sometimes this deposit is so extensive as to spread over the entire fœtal superficies. These effusions are observed in placentas expelled at the full term of gestation, the children being born alive and

healthy. They do not appear to impede the circulation through the placental vessels; but in some cases I have witnessed, the quantity of effused fibrine was so great in the proximity of the large vessels gathering to constitute the umbilical trunks, that I have been surprised that these vessels have escaped pressure, or even obliteration, from the great contraction to which effused fibrine is liable. In connection with the form of effused fibrine to which I am now referring, I have not detected any evidence of inflammatory action. The placental tissue in the immediate vicinity has been perfectly normal. I am disposed to believe that fibrine may be poured out into the placenta as the simple consequence of that excessive proportion of fibrine which is characteristic of the blood during pregnancy.

In concluding, I may be permitted to remark that the carefully-observed facts related in this and my former paper, tend to prove that fatty degeneration of the placenta is far from being an uncommon event. That it has hitherto escaped observation is to be attributed to the neglect of the microscope in investigating the abnormal conditions of that organ. Alterations of structure in the elementary tissues of the placenta cannot be recognised without the light of that instrument; and, on the other hand, the gross appearances to the naked eye frequently lead to the most erroneous conclusions. I have already remarked, that placentas have been submitted to me as examples of fatty degeneration which, under the microscope, exhibited the villi in a perfectly healthy state. On the other hand, I confidently believe that the real alteration in many placentas, described by various authors as examples of scirrhous disease, of atrophy, or of tubercle, might have been shown to be fatty degeneration, had the morbid portions been subjected to microscopical analysis.

Postscript, *Feb.* 16, 1853.

Case xii.—I beg permission to report yet another Case which has occurred since my paper was placed in the hands of the Secretary. It serves to fill up what many may consider to have been a void in the evidence supplied by the cases hitherto reported.

On the 6th February, 1853, a stout adipose woman was delivered of a healthy child, strong and of average size, after a natural labour. She is a patient of the Paddington Infirmary. Dr. Mackenzie had caused the placenta to be examined, because he had, on a former occasion, observed a fatty placenta in a woman who was unusually stout and inclined to obesity.

The *placenta* was above the average size; a great proportion appeared healthy. Along the circumference was a thick dense fibrinous-looking deposit. There were three or four nodules or masses of degenerated placenta of the size of a walnut on the maternal surface. One of these nodules, especially examined, was of a glistening whitish-yellow aspect, and exsanguine. Under the microscope it was found to consist entirely of villi undergoing granular degeneration; in parts the granules or spherules of oil were numerous, and the chorion investing the vessels more or less broken up. The villi in the vicinity of this nodule, which to the naked eye appeared healthy, were also affected, but to a less extent. The villi in other parts were healthy.

It seems difficult to attribute the commencing degeneration of the placenta in this case to mal-nutrition, or to any other state of the fœtus, which was born alive and strong. The alteration was limited in extent, and no doubt commenced late in pregnancy. Had it begun at an earlier period, it might have had time to invade a large portion of the organ, and have cut off the child before the natural epoch of parturition.

This case appears to me to prove conclusively that fatty degeneration of the placenta may arise independently of

death or disease of the embryo. This point established, it is unnecessary to show that when this degradation of structure has invaded a large proportion of the placenta, the organ must be rendered unfit for the performance of its functions, and that the fœtus must perish in consequence.

This case supplies the last link that was wanting in the chain of evidence, to confirm the leading propositions I have advanced in reference to the pathological value of fatty degeneration of the placenta.

RESEARCHES ON THE

PATHOLOGY OF OBSTRUCTIVE PHLEBITIS,

AND

THE NATURE AND PROXIMATE CAUSE

OF

PHLEGMASIA DOLENS.

BY

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SINCE the publication of the admirable paper of the late Professor Davis, in the 12th volume of the 'Transactions' of this Society, the profession has very generally recognised the important relations which subsist between certain lesions of the crural veins and the general phenomena of phlegmasia dolens. The facts brought forward by Dr. Davis have been fully substantiated by the investigations of various pathologists, and no doubt can be said to exist at the present day as to their accuracy or authenticity. With regard, however, to the conclusions which have been drawn from them, there is less unanimity of opinion; and whilst many, concurring with Dr. Davis, consider the affection of the veins to be the proximate cause of the disease, others believe it to be of secondary importance, or an effect rather than the cause of the disease. It is the design of the following communication to inquire more particularly into the correctness of these respective opinions,—to determine more specifically than has hitherto been done, the pathology of obstructive phlebitis; and, if

possible, to assign its true relations to the other phenomena of the disease.

The characteristics of phlegmasia dolens are, in the words of Callisen, "tumor elasticus, albescens, renitens, calidus, dolens, foveam impressi digiti haud retinens." It is described by Dr. Hull as consisting in a "tense elastic hot painful swelling, which generally extends rapidly over the whole of one of the lower extremities; the skin retaining its natural colour, or even becoming whiter, and presenting more or less of a shining appearance."

In its local manifestations it would thus appear to be characterised by two principal conditions:

1st. By a persistent and peculiar swelling of the affected limb not depending upon simple œdema, but possessing a degree of tension, heat, firmness, and elasticity, which is not common to ordinary œdema, and

2dly. By an impairment of the nervous and muscular functions of the limb, as indicated by pain, tenderness, and loss of motor power.

I will proceed, in the first place, to consider how far these conditions are producible by inflammation of the pelvic or crural veins, by comparing them with those which follow upon such inflammation artificially induced.

To determine this question I had recourse to the following series of experiments, in which I was kindly assisted by Mr. Briggs, Demonstrator of Anatomy at University College, whose valuable services I cannot sufficiently acknowledge. First, to ligaturing the iliac veins, and observing the resulting phenomena at definite periods after the operation. Secondly, to extensive irritation of the lining membrane of the iliac veins by means of chemical and mechanical irritants. Thirdly, to sustained compression of the femoral veins by means of firm metallic compresses. I will proceed, in the first place, to give the results of ligaturing the iliac veins, as noted at periods of 24, 48, 72, 96 hours, and nine days after the operation.

EXPERIMENT I.—The right common iliac vein of a dog was ligatured on the 26th November, 1851, and in twenty-four hours the animal was killed and the parts immediately examined. For some hours after the operation he remained quiet and refused food, but before the expiration of the twenty-four hours he had greatly recovered, took food, and was able to walk about. There had been much swelling of the ligatured extremity subsequently to the operation, but this was inelastic, and readily pitted on pressure.

Dissection.—There was considerable inflammation of the wound, by which the vein had been exposed; and the cellular tissue throughout the limb was infiltrated with serum, as was also the adjoining part of the right side of the abdomen. In the neighbourhood of the wound, the abdominal muscles were soft, swollen, and œdematous. The common iliac vein was empty above the ligature; but below it, there was a soft, dark-coloured coagulum, of about two inches in length. It was no where adherent to the vein, nor did it possess any other characters than those which are common to an ordinary post-mortem coagulum. The vein at the seat of ligature was slightly thickened and somewhat opaque; and on its interior a few small vessels could be seen, immediately where the ligature had been applied. Beyond this there was no other evidence of inflammation; and the lining membrane of the vein was smooth, and contained neither pus, lymph, nor any other inflammatory product. The morbid appearances were confined to the immediate seat of the ligature, and did not extend more than half an inch above and an inch below it. The vena cava was healthy, as were also the femoral vein and its branches on the right side, and the pelvic and femoral veins generally on the left.

EXPERIMENT II.—The right common iliac vein of a dog was ligatured on the 4th December, 1851, and the animal was killed and the parts examined exactly forty-eight hours afterwards. For some hours after the operation he refused food, remained quiet, and walked with extreme difficulty. There had also been considerable œdema of the affected

extremity, but this was soft and inelastic. In thirty-six hours the swelling had somewhat subsided, and the dog had generally recovered from the effects of the operation. He took food and was able to walk about.

Dissection.—The limb generally was œdematous, and there was considerable inflammation of the wound, by which the vein had been exposed. The abdominal muscles around were œdematous, and the intermuscular cellular tissue contained some purulent looking matter. The iliac vein both above and below the ligature was adherent to the surrounding tissues. The portion above the ligature was empty; that below contained a soft uniformly black coagulum, of about 2 inches in length, which was no where adherent. On removing it the inner surface of the vein was found to be morbidly vascular at the seat of ligature. Here also the vein was thickened and opaque, and its coats were partially divided by ulceration. There was no lymph or purulent matter anywhere observable in it, and the morbid appearances terminated at little more than an inch both above and below the ligature. The other pelvic veins, the femorals and the vena cava, were healthy.

Experiment iii.—The left common iliac vein of a dog was ligatured on 1st December, 1851, and the animal was killed and the parts examined seventy-two hours afterwards. The immediate effects of the operation were similar to those which occurred in the preceding cases. Considerable swelling of the extremity took place, of a soft inelastic character, and during the first day he remained quiet and refused food. The following day he was better, ate well, and was able to walk about; and from this time the swelling gradually subsided, so that very little was perceptible on the third day subsequent to the operation.

Dissection.—Considerable inflammation had taken place throughout the wound, by which the vein had been exposed. The subcutaneous vessels of the upper part of the thigh, and those of the corresponding side of the abdomen, were much injected, and the subcutaneous cellular tissue of the limb

contained a small quantity of serum. The muscles of the left side of the abdomen were swollen, and the intermuscular cellular tissue contained some purulent-looking matter. The left common iliac vein was nearly ulcerated through at the seat of ligature. The vein above the ligature was empty, but the portion below contained some coagulated blood, which extended nearly to the femoral vein, and was of a uniformly black colour, soft consistence, and could be easily removed. The vein below was morbidly vascular, and almost uniformly red; this, however, was partially the effect of staining. The vein was thickened and opaque at the seat of ligature, but throughout its interior there was no fibrinous, purulent, or other inflammatory product. The morbid appearances terminated about two inches above and below the ligature. The vena cava was healthy and empty, as were also the other veins generally.

EXPERIMENT IV.—The right common iliac vein of a dog was ligatured on the 19th December, 1851, and the animal was killed and the parts examined exactly ninety-six hours afterwards. The dog at first was quiet and disinclined for food; but after the first day ate well, and was able to walk about. At first there was considerable œdema of the limb, of a soft inelastic character, but this gradually subsided after the first day.

Dissection.—The wound through which the vein had been exposed, had in a great measure contracted. The tissues around the vein were found to have been considerably inflamed, and the abdominal muscles and intermuscular cellular tissue about the wound were still somewhat infiltrated. Very little serum was found in the cellular tissue of the affected limb. The iliac vein was completely divided at the seat of ligature, and the divided extremities were much thickened, morbidly vascular, and closed by adhesive inflammation. The upper portion of the divided vein was empty, the lower was closed by a coagulum which extended downwards for about an inch and a half. This coagulum was closely adherent to the vein at the point of its division, but below the

adhesion was less intimate, so that it could be easily separated. No puriform matter was observable in the vein.

Experiment v.—The left common iliac vein of a dog was exposed and ligatured on the morning of the 11th October, 1851, and it was killed on the 20th, nine days afterwards. On the day of the operation the dog was sensibly affected, refused food, and moved with difficulty; but the next day he was better, and from that time continued to improve. Considerable swelling of the limb followed the application of the ligature, but this began to subside the day after, and in the course of a few days was scarcely perceptible. It was throughout soft and inelastic.

Dissection.—The wound through which the vein had been exposed, had almost entirely closed. There was little or no serum perceptible in the cellular tissue of the limb. The vein was found to have been completely divided by the ligature, and a considerable quantity of blood had escaped from it into the surrounding cellular tissue, which extended upwards as high as the left kidney. It was of a dark colour and grumous consistence. The ligatured vein had contracted intimate adhesions to the surrounding parts. Its coats were much thickened about the seat of ligature, and its divided edges were somewhat tuberculated, and of a violet colour. The upper portion of the vein was closed at the point of division by adhesive inflammation, and its calibre was somewhat contracted. It was empty, and greatly thickened. The lower portion of the vein was also much thickened, and of a violet colour immediately below the ligature. Within it fibrine had been deposited, which was so adherent that it was scarcely possible to detach some portions of it. The morbid appearances of the vein were limited to the space of about an inch and a half below the ligature. The vessel contained neither pus nor a coagulum, and although its colour was of a deep red or violet tint, no distinct vascularity could be observed.

The foregoing particulars may be said to comprehend the

more important phenomena which follow upon ligature of the iliac veins, and afford some data for comparing the effects of inflammation of these vessels thus induced with the general symptoms of phlegmasia dolens, and the comparison will perhaps be assisted by the following summary of the principal facts which were observed:

The constitutional effects which follow upon ligature of the iliac veins, would appear to be of a very transient character, and to be rather referable to the extensive wound which is necessary to expose the vein, than to the actual operation itself. They were most marked and severe on the day of the operation, during which the animal remained quiet, subdued, and disinclined to take food. At the end of twenty-four hours they had in a great measure subsided, and from this time almost rapidly disappeared. The local effects were also of a transient rather than of a persistent character. They were confined in a great measure to swelling, stiffness, and an inability or disinclination to move the affected extremity. For, as far as could be judged, there was neither tenderness on pressure, nor any preternatural heat of the limb, except in the immediate neighbourhood of the wound. These symptoms, moreover, were not of long continuance. The swelling was greatest during the first and second days following the operation, and it then steadily and quickly disappeared. Its character, again, was that of ordinary œdema—it readily pitted on pressure, and was nearly equally diffused over the ligatured extremity.

The morbid condition of the ligatured veins may be thus recapitulated. At the end of twenty-four hours after ligature, the coats of the vein were slightly thickened, and opaque at the immediate seat of ligature, and a loose, thick, unadherent coagulum, of about two inches in length, occupied the interior of the vein, immediately below it. In forty-eight hours there was increased opacity, thickening, and vascularity of the vein at the seat of ligature, and these appearances extended for about an inch above and an inch and a half below it. Ulceration of the coats of the vein had now commenced, and the vessel had contracted ad-

varices: Œtius, Paulus, Avicenna, and Albucasis describe the operation of excision. This seems to have been the practice of Fallopius and Severinus. Fabricius Aquapendens and Fabricius Hildanus inclosed the varicose vein between two ligatures, and emptied it by incision. Ambrose Paré, Petit, Dionis, and others, emptied it by punctures and brought its sides into contact by compression. . . . . Many of the older operating surgeons in the country, army and navy, still adopt the practice of tying veins after amputation, and are unwilling to believe that it can ever be productive of mischief. I have frequently," says Mr. Travers, " seen the femoral vein tied without any obvious ill effect; and one of the most experienced and successful operators in the West of England lately assured me that he had been in the constant practice of tying the main vein distinctly from the artery in amputation. Neither do the Continental surgeons entertain any apprehension of a ligature upon a vein, nor are they, I believe, in any degree aware of the extensive inflammation to which the interior tunic of these vessels is in consequence exposed." The experiments I have related fully attest the accuracy and justness of these remarks, and it will be sufficiently shown in the sequel that operations upon veins are not in themselves productive of either serious or dangerous consequences, and that the formidable symptoms which sometimes follow upon such operations, have another origin, and may be traced to a very different cause.

I proceed, in the next place, to detail the results of more extensive irritation of the iliac veins, as observed in the following experiments. In the first place, a strong solution of the nitrate of silver was applied to the whole extent of their lining membrane. In the second, a solid piece of bougie was passed into them, so as to occupy their interior, and therein secured for several days.

Experiment vi.—The right femoral vein of a dog was ligatured on the 8th of June, 1852; and an opening having been made in it above the ligature, a solution of the nitrate of silver (℈ij to ℥j) was freely introduced upon lint, and

applied to the whole interior of the corresponding iliac veins. The opening in the femoral vein was now closed by ligature, and the effects of the operation were carefully watched for several consecutive days.

In 24 hours there was very little constitutional disturbance. The leg was somewhat swollen; but it could be moved without any apparent difficulty, and the swelling was soft and inelastic. There was no particular tenderness of the limb, nor was there any indication of the inflammation having extended to the femoral vein. In 48 hours the appearances were much the same: the inflammation had not extended to the femoral vein, nor was there any increased swelling or loss of motor power. In 72 hours no fresh symptoms had developed themselves; on the other hand, the swelling and stiffness had began to subside, and the dog appeared to be otherwise perfectly well. It was allowed to live for ten days subsequently to the operation, but without any fresh symptoms occurring in connection with the operation.

Dissection.—On tracing the femoral vein upwards, from the opening which had been made into it, it was found that the external and common iliac veins were filled with a firm coagulum to within half an inch of the cava, forming a solid, corded body. On opening them, it was found that this coagulum was firmly adherent to their lining membrane, and was for the most part of a yellowish-brown colour, except at its superior extremity, where it was deep red. Its central portion had begun to soften down, and was of a semi-pultaceous consistence; but its exterior was firm, and adhered closely to the vein. The coats of the vein were generally thickened, and externally vascular; and on removing the coagulum, the lining membrane was left roughened, patchy, and opaque. The femoral vein below the ligature was healthy, pervious, and empty. The inflammation had not extended beyond the irritated portion of the veins. There was no œdema of the extremity or any other morbid appearances met with, which were attributable to the operation.

EXPERIMENT VII.—On the subsidence of the local symptoms in this case, the left femoral vein was opened, and a solid piece of bougie, about five inches long, was passed into the left external and common iliac veins as far as the cava, and therein secured by means of a ligature. The effects of this operation were carefully noted for several days, and may be thus recapitulated:—24 hours afterwards the limb was but little swollen, and scarcely at all painful; the dog had eaten food, and was not perceptibly the worse for the operation. It could stand, walk, and even run without any apparent difficulty. In 72 hours the swelling had disappeared, and the dog was not otherwise worse than on the preceding day. No fresh symptoms developed themselves; and five days subsequently to the operation the dog was killed, and the parts immediately examined.

Dissection.—The bougie was found to have occupied the entire length of the external and common iliac veins, and to have extended about two and a half inches into the vena cava. It had become softened down in places, and was covered with a thin layer of coagulum. It was adherent to the track of vein with which it was in contact, but could be easily detached; and on removing it, the lining membrane was left smooth, free from redness or vascularity, and apparently perfectly healthy. No other morbid appearances were observed throughout the veins of this extremity, except a little thickening and obstruction of the femoral at the point at which it had been ligatured.

I shall conclude this part of the subject by detailing the particulars of a case in which prolonged irritation and compression of the femoral vein was maintained by means of a firm metallic compress.

EXPERIMENT VIII.—The right femoral vein of a dog was exposed on the 10th of December, 1851, and a strong metallic compress was secured upon it, so as to maintain firm compression of its coats, and at the same time to arrest the circulation through it. It was kept on for six hours,

and then removed. No perceptible swelling of the limb, or loss of motor power, followed the operation; and after examining the limb daily until the 16th, without any morbid appearances having been perceived, the dog was killed, and the vein examined.

Dissection.—The limb presented no œdema, or any other abnormal appearance. The coats of the vein were thickened, indurated, and opaque at the part at which the compress had been applied; but there was no redness or morbid vascularity. These appearances were limited to about half an inch of the extent of the vein. A small thin coagulum was found in this part of the vessel, of about an inch and a half in length; but it was nowhere adherent, and the vein was otherwise perfectly healthy.

We are now, I think, in a position to appreciate the full force and influence of inflammation of the iliac and femoral veins in the causation of the symptoms of phlegmasia dolens, or rather, to speak more specifically, in the causation of a general, abiding, and elastic swelling of the affected limb, attended with an impairment of its sensory and motor powers; and the conclusion, I submit, must inevitably be, if the foregoing experiments possess any value or reality, that such symptoms are not producible by mere inflammation and obstruction of these vessels, however rapidly induced. Thus, as the result of obstruction, inflammation, ulceration, and total division of the common iliac vein, produced by the application of the ligature, we fail to observe more than the necessary effects of mechanical and temporary obstruction of the vessel, and of inflammation of it, limited to the immediate seat of ligature. There is an absence of any constitutional fever or disturbance; the swelling of the limb is neither elastic nor abiding, but simply œdematous, and there is no impairment of either its sensory or motor functions. With such facts, clearly demonstrated, there is, I submit, no other conclusion than that the affection of the veins is not the sole or essential lesion, or the proximate cause of the complaint.

But whilst I believe that this conclusion is not only deducible from the foregoing experiments, but will be found upon the whole to be supported by the clinical history of phlegmasia dolens, it must ever be remembered that an inflamed and obstructed condition of the iliac veins constitutes one of the most important and indeed pathognomonic features of the complaint; and therefore, in seeking for a theory of the disease, we must exclude any which does not equally account for this as well as its other pathological characters. The problem, then, which has to be solved is, what are the specific causes or conditions which can simultaneously affect the sensory, the motor, the secretory, and the vascular organs of the affected extremity, so as to give rise to the concurrent phenomena by which the disease is characterised. Are they local or constitutional? and in what do they respectively consist? Now, these are questions which cannot be satisfactorily answered until we have correctly determined the pathology of *obstructive* phlebitis, and more particularly to what extent it is dependent upon local as distinguished from constitutional causes.

I have accordingly pursued the investigation of this subject at some length, from a conviction that it was absolutely necessary to a correct appreciation of the pathology of phlegmasia dolens; for, if we look to standard works or to recognised authorities for information, we shall find very different, and indeed contradictory, opinions expressed in regard to it. Thus, by the majority of writers, the phenomena in question have been attributed generally to phlebitis. But phlebitis in a variety of ways may and does constantly occur without giving rise to it. John Hunter, for instance, informs us that in all violent inflammations of the cellular membrane, whether spontaneous or in consequence of accident, as in compound fractures, or of surgical operations, as in the removal of an extremity, that the coats of the larger veins passing through the inflamed part become also considerably inflamed. Again, in the numerous operations which have been performed upon the veins for the cure of varix, varicocele, and other morbid

conditions, including ligature, excision, and cauterisation, it is certain, that while venous inflammation must ensue from their performance, such inflammation is not ordinarily productive of any serious or extensive obstruction of these vessels. I submit, then, that the theory of phlebitis, as the supposed cause of such obstruction, is not only vague, but in some degree inaccurate—inasmuch as it is not mere phlebitis which gives rise to it, but something peculiar in or superadded to it. Phlebitis, as I have remarked, may occur in a variety of ways without occasioning it; and, therefore, it is less phlebitis than these peculiar or superadded circumstances which should more especially claim our attention.[1]

Again the phenomena of *obstructive* phlebitis have been attributed to causes acting primarily upon the blood, and to retardation or coagulation of this fluid in the veins, either from extreme vital exhaustion, or the direct action of diseased secretions upon it, and here it is assumed that phlebitis, if at all produced, is secondary to or consequent upon such primary coagulation of the blood. Between these opposite, and it may be said contradictory, opinions, it is difficult to decide, if we are guided solely by authority, or the weight of individual testimony. I have therefore pursued the investigation of this subject irrespectively of

[1] As the use of the term phlebitis, in different senses, may appear to give rise to some ambiguity in the text, I would beg to observe, that it may be regarded as applying generically to three distinct species or varieties of venous inflammation—the *healthy*, the *obstructive*, and the *suppurative*. The first occurs on the reparation of all wounds, injuries, and operations upon veins, is of limited extent, and never productive of any serious or dangerous consequences—indeed, medical men practically act upon this conviction in the daily exercise of their profession. In certain rare or exceptional cases, however, operations, &c. upon veins are followed by extensive inflammation of their vessels of an obstructive or suppurative character, and the question arises, what are the causes of such peculiarities? how far do they depend purely upon inflammation of the coats of the veins? and how far upon other causes? It is this question, or rather that part of it, which relates to *obstructive* phlebitis, that I have more especially endeavoured to elucidate in the present inquiry.

either, and have more especially endeavoured to ascertain the respective influence of the blood and the veins, as well as their conjoint operation in its causation. Following, then, this plan of inquiry, I shall proceed to give in detail the results of irritating and otherwise injuring the external and the internal coat of veins; of such proceedings as were calculated to affect primarily the blood; and of such as were calculated to elicit the nature of their combined influence in giving rise to the phenomena in question.

Of Irritation and Injury of the External Coat of Veins, as connected with the causation of Obstructive Phlebitis.

To determine the nature of this influence, I had recourse to the following experiments, in all of which the external coat of veins was either mechanically injured or irritated by the free application of some corrosive or chemical irritant.

Experiment ix.—The right jugular vein of a dog was exposed for about three inches, on the 21st of January, 1852, and its sheath having been opened, a solution of the nitrate of silver (℈ij to ʒj) was brushed several times over it, to the extent of about 2 inches. The dog continued perfectly well until the 23d, when it was killed, and the parts immediately examined.

Dissection.—The wound throughout was greatly inflamed. The tissues around the vein were morbidly vascular, and matted together by adhesive inflammation. The vein itself was contracted; and the portion to which the solution had been applied, was coated with a fibrinous looking deposit, and adhered closely to the surrounding tissues. The coats of the vein were here also somewhat thickened and reddened. On opening the vein it was found to be perfectly pervious, and to contain only a thin thread of coagulum, which was adherent at its superior extremity. The lining membrane was tinted with red striæ or spots, but no distinct vascularity could be seen; and between these striæ,

the lining membrane had an opaque blueish appearance. The coats of the vein were here also somewhat thickened. These appearances were strictly limited to the portion of vein which had been irritated, above and below which it was perfectly healthy.

EXPERIMENT X.—The right jugular vein of a dog was exposed for about three inches, on the 17th January, 1852, and its cellular sheath having been opened, the vein was lightly brushed two or three times over with a stick of lunar caustic. The dog remained well until the 19th, when it was killed, and the parts examined.

Dissection.—On opening the wound by which the vein had been exposed, it was found to be filled with coagulated blood. The portion of vein to which the nitrate of silver had been applied, had sloughed away, and within its interior there was a thin thready black coagulum, which was no where adherent. The coats of this portion of the vein were much thickened, and its calibre contracted.

On removing the coagulum, the interior of the vein had an opaque appearance, and was slightly reddened; but no fibrine or lymph had been anywhere exuded. The morbid appearances were limited to the injured portion of the vein, and both above and below this it was perfectly healthy and pervious.

EXPERIMENT XI.—The right jugular vein of a dog was exposed, on the 26th January, 1852, and its sheath having been opened, it was forcibly pinched and contused in several places. The wound was then closed by suture, and in forty-eight hours the dog was killed, and the parts examined.

Dissection.—The wound throughout was much inflamed, and the portion of vein which had been injured was covered with a fibrinous-looking matter which was adherent, but could be readily detached. This portion of the vein was somewhat contracted, but was pervious, and on opening it, it was found to be empty, and free from any coagulum or

deposit of any kind; indeed, the lining membrane may be said to have been perfectly healthy.

EXPERIMENT XII.—The right jugular vein of a dog was exposed for about three inches, on the 19th January, 1852. It was then ligatured in two places, intermediately opened, contused, and again ligatured. The dog continued well for forty-eight hours, and was then killed, and the parts examined.

Dissection.—The wound through which the vein had been exposed was found filled with coagulated blood, which had evidently escaped from the vein. The tissues around it were much inflamed, and the vein itself was adherent to them, and covered with a fibrinous looking matter. On removing this, the exterior of the vein was found to be morbidly vascular, and its coats thickened. On opening it, it was empty, but immediately below the superior or distal ligature there was a small unadherent coagulum of about half an inch in length. The lining membrane was smooth, except inferiorly, where the vein had been contused.

Here and there, there was some little vascularity, especially where the vein had been opened and the ligatures applied, but there was neither lymph, fibrine, or pus anywhere perceptible, and the morbid appearances were strictly limited to the injured portion of the vein.

From the facts elicited from the foregoing experiments, the following inferences appear to be deducible:

1st. That obstruction of veins is not a necessary consequence of all forms of venous inflammation, and in particular of that which follows upon irritation or injury of their external coat.

2dly. That inflammation of veins thus excited is not disposed in a healthy animal to extend itself indefinitely, but on the other hand is strictly limited to the immediate seat of such irritation or injury.

3dly. That the external coat of veins very readily reacts under the influence of irritating causes; this being, in these cases, morbidly vascular, covered with inflammatory lymph, and adherent to the surrounding tissues.

4thly. That such reaction, followed by considerable inflammation of the external coat, may occur without giving rise to any corresponding inflammation of the lining membrane: for, in these cases the latter was healthy, and the vein consequently pervious, or at least free from any inflammatory exudation.

And if we admit the accuracy of these conclusions, it must follow: that neither operations upon veins, such as ligature, excision, or division, nor rheumatic inflammation of their external coat, nor mechanical injuries, such as contusion or laceration, are essentially the causes of obstructive phlebitis, as has sometimes been supposed. Nor is it probable that irritation or injury of the orifices of the uterine veins, whether occasioned by instrumental operations or the forcible separation of the placenta, can excite inflammation or obstruction of the crural veins, by being propagated to them along the coats of the hypogastric; for we have seen that, in a state of heath, inflammation of veins consequent upon mechanical injury is not so disposed to extend itself. Lastly, inasmuch as it would appear that the external coat of veins very readily reacts under the influence of irritating causes, we may conclude that irritation of their lining membrane may, in some cases, give rise to it, and so to those general changes in the consistence, opacity, and vascularity of the vein, which are ordinarily regarded as characteristic of phlebitis.

Of Irritation and Injury of the Internal Coat of Veins as connected with the causation of Obstructive Phlebitis.

The phenomena which follow upon irritation of the lining membrane of veins have been very differently described by

different pathologists, although in themselves they are sufficiently simple and uniform. When, for instance, an irritant is thrown into a vein, the almost invariable consequences are, obstruction of the vessel, stagnation and coagulation of the contained blood, and sooner or later, thickening, opacity, and increased vascularity of its coats. But the particular manner in which these phenomena take place, have been differently represented by different observers. Thus by some it is maintained that phlebitis is primarily induced, and that this gives rise to the effusion of coagulable lymph into the vein, and to coagulation of the contained blood. By others, however, it is considered that the action of the irritant is primarily upon the blood, and that coagulation of fluid precedes and gives rise to the phenomena of phlebitis.

To determine the correctness of these opinions, it is necessary to investigate, separately, the effects of irritating the lining membrane of veins under the circumstances of the blood being permanently excluded from, as well as subsequently readmitted into, the irritated vein, for without doing so it is impossible to distinguish between the share respectively taken by the blood and the vein in the causation of any consequent phenomena. I propose, therefore, to confine myself, in the first place, to a consideration of the effects of irritating the lining membrane of veins under the circumstance of the blood being permanently excluded, and will subsequently treat of the effects which follow upon such irritation, when the blood has been subsequently readmitted into the irritated vein.

Although it has been generally held, that the veins are not very susceptible of inflammation from mechanical injury, it is yet believed, that they are susceptible of rapid inflammation when irritating fluids are applied to their lining membrane, and that consequent upon this a plastic material is effused from the latter, which together with coagula derived from the blood, constitutes the material by which veins are obstructed, and eventually obliterated under these circumstances. This at least is the doctrine propounded by Hasse in his excellent treatise on 'Pathological Anatomy,'

and he quotes the following experiment by Gendrin in support of it. Having secured a portion of an artery between two ligatures, and entirely cleansed it of blood, this experimenter states that he discovered, after throwing in an irritant injection, a plastic substance deposited within the part so insulated, filling up the whole calibre of the vessel; and that similar trials with veins led to the same results. "Hence it is natural to infer," says Hasse, "that in vessels containing blood the plastic product partly exudes from the parietes of the vessels, and is partly deposited from the blood." Now, with reference to these remarks, I would observe, that in several instances in which I repeated very similar experiments to those described by Gendrin, the results were different; as shown in the following:

EXPERIMENT XIII.—The right jugular vein of a dog was exposed for about three inches, on the 15th January, 1852, and the exposed vein was included between two ligatures, so as to leave about two inches of it between them. An opening was now made in the included portion, and some tepid water having been injected for the purpose of washing away any blood, a solution of the bichloride of mercury (gr. iij to ʒj), was injected into it, and again removed after having been allowed to remain in about half a minute. In the operation some of the injection came in contact with the exterior of the vein and surrounding parts, but this was speedily wiped away. The dog appeared to be but little affected by the operation, and continued well until the 17th, when it was killed, and the vein examined.

Dissection.—The wound throughout was much inflamed. The vein adhered closely to the surrounding parts, especially where the injection had come in contact with it externally. Here also the exterior of the vein was covered with adherent lymph, which gave it a thickened, roughened, solidified appearance. On removing this, the outer coat of the vein was found to be of a deep red colour, and morbidly vascular. The vein itself was contracted and apparently empty, but

on opening it, a thin thread of coagulum was found in it, of a dark red colour, soft consistence, and nowhere adherent. The lining membrane was of a uniform dingy red colour, but was smooth, and free from lymph, fibrine, or pus. Its texture was good, and it was firm, elastic, and extensible. On the proximal side of the ligatured part the vein assumed a healthy character, immediately below the point which had been injected. The included part contained a small soft coagulum. At the seat of each ligature, the coats of the vein were somewhat thickened and vascular, but beyond, they were perfectly healthy in each direction.

Experiment xiv.—The jugular vein of a dog was exposed for about three inches, on the morning of 10th January, 1852, and after it had been emptied of blood, about two inches of it was included between two ligatures. An opening was then made in this portion, and a solution of the sulphate of zinc (gr. iij to ʒj), was injected into it. It was allowed to remain in for about two minutes, and then withdrawn, and the proximal ligature removed. The dog was very little affected by the operation, and was killed, and examined forty-eight hours afterwards.

Dissection.—There was considerable inflammation throughout the wound. The tissues around the vein were morbidly vascular, infiltrated, and matted together by adhesive inflammation. The exterior of the vein was covered with a fibrinous looking deposit, having a semitransparent appearance, and of a red colour. It adhered throughout to the vein, but could be easily removed; and on removing it the external coat of the vein was found to be dotted with vascular points. The portion of the vein which had been injected was somewhat shrunk and diminished in calibre, and its coats were thickened and opaque. On opening it, it was found to be empty, and the lining membrane had a pinkish-violet tint, with here and there distinct vascularity.

This vascularity was most apparent at the point at which the ligatures had been applied, and at the part which had

been opened for injection. Throughout this portion of the vein the lining membrane was perfectly smooth, and free from any lymph or fibrinous deposit. A small coagulum of about half an inch in length was found on the proximal side of the opening which had been made in the vein, and one of about a quarter of an inch in length on its distal side. The morbid appearances were strictly limited to the injected portion of the vein, beyond which it was perfectly healthy, in each direction.

EXPERIMENT XV.—The right jugular vein of a dog was exposed for about three inches, on the 19th January, 1852, and its sheath having been opened, two ligatures were applied, so as to include about two inches of it between them. This was then emptied of blood, and a solution of the nitrate of silver (℈ij to ʒj) was freely passed into its interior, on lint. The dog did not appear to be sensibly affected by the operation, and continued well for three days, when it was killed, and the parts immediately examined.

Dissection.—The wound by which the vein had been exposed, was throughout much inflamed, and filled with coagulated blood. The vein had been completely divided by the distal ligature, and at the point of division its coats were much thickened and tumefied. Externally, the portion of vein which had been irritated was covered with inflammatory lymph, and was adherent to the surrounding tissues, and here also its coats were much thickened. The interior of this part of the vein was almost perfectly empty. It contained no coagula, and its lining membrane, although somewhat reddened and patchy, was apparently free from any lymph which had been exuded from the lining membrane.

The foregoing experiments having been performed solely for the purpose of determining the effect of irritation of the lining membrane of veins, in the absence of the blood, I will merely remark, that they are completely at variance with the observations of Mons. Gendrin,

as to the dependence of obstruction of veins upon the effusion of coagulable lymph from their lining membrane excited by inflammation. For in these cases it would appear, notwithstanding the application of such irritants to this membrane, as solutions of the bichloride of mercury, sulphate of zinc, and nitrate of silver, that the veins so treated remained either perfectly empty, or contained only some fibrinous looking matter, having no analogy to coagulable lymph, and evidently deposited upon, rather than exuded from, the vein. We cannot then suppose, that veins are ordinarily obstructed, by the effusion of such lymph, although it is far from my intention to affirm that this never takes place. On the other hand, I have fully ascertained that under certain circumstances lymph is actually exuded from the lining membrane of veins, but so far as I can judge, its tendency is to develope suppurative rather than obstructive phlebitis; and restricting my observations to the latter, I feel justified in affirming, upon the strength of the experiments I have related, that considerable irritation of the lining membrane of veins may be excited without giving rise to the effusion of any appreciable amount of coagulable lymph.

And, as bearing upon the general pathology of phlebitis, it should be particularly remarked, that in all these cases the morbid appearances were strictly limited to the irritated and injured portion of the vein. Beyond this it was perfectly healthy, pervious, and free from coagula; and in the application of this fact to the pathology of the veins, we may learn that artificial irritation of their lining membrane, as of their external coat, is not disposed to extend its effects beyond the original seat of irritation or injury, and consequently, in those cases in which the veins are extensively obstructed, as is the case in phlegmasia dolens, that the exciting cause of such obstruction must have been applied coextensively with the obstruction itself.

The origin of obstructive phlebitis from spontaneous coagulation of blood in the veins, may be considered under two heads:—first, coagulation of blood in these vessels from

extreme physical or vital prostration; and, secondly, coagulation of this fluid from the direct action of morbid matters upon it. I proceed, in the first place, to treat of the former, and will subsequently consider the latter.

The doctrine of obstruction of veins from spontaneous coagulation of blood in them, consequent upon extreme vital exhaustion, is one which derives some support from the circumstances by which phlegmasia dolens is sometimes preceded. Thus the attack has often followed upon profuse losses of blood, is frequently ushered in with symptoms of feebleness and prostration, and occurs for the most part in persons who had been previously in a depressed state of health. But that these circumstances are not specifically its cause, is proved by the fact that it has occurred in persons possessed of much strength and vigour of constitution, and has been successfully treated by bloodletting and depletion. However, to test the question by direct experiment, I had dogs repeatedly bled and kept low, but without observing any consecutive obstruction of the veins to ensue. I had then recourse to ligature and compression of these vessels, after such depletion had been practised, but still no extensive coagulation of blood took place beyond the seat of such ligature or compression. On the other hand, there was less in these cases than in those in which no such depletion had been practised. These experiments need not be further particularised, as they were upon the whole almost uniform in their results; but the two following are, on other grounds, worthy of attention. The first illustrates the effects of the ligature of veins in a parturient animal, after copious depletion. The second, the effects of severe and extensive injury of veins together with considerable loss of blood.

Experiment xvi.—The right femoral vein of a bitch, which had littered on the 14th April, 1852, was ligatured on the 16th of the same month, a large quantity of blood having been allowed to escape during the operation. On the 17th there was no swelling, and scarcely any lameness

of the affected extremity. On the 19th, matters being the same, the left femoral vein was tied close to the iliac, and this was followed by distension of the superficial veins of the limb. On the 20th there was some lameness, but no swelling, and on the 21st, as nothing further was perceptible, she was again bled to the extent of ʒiij. The limb was examined for several consecutive days, but no swelling or stiffness could be discovered, nor was there any particular obstruction of the ligatured veins. After some days had elapsed, the animal was killed, and on examination a little induration and consolidation of the femoral veins were alone found at the immediate seat of the ligatures.

Experiment xvii.—The right femoral vein was exposed on the morning of the 19th December, 1851, and ʒiij of blood having been taken away from a small artery in the neighbourhood, a strong elastic metallic compress was applied round the vein, and secured on it by ligature. This had the effect of compressing and contusing the coats of the vein, while at the same time it arrested the flow of blood through it. It was continued on for forty-eight hours, during which period the dog was not perceptibly affected, nor was there any particular swelling or stiffness of the corresponding extremity. It was now removed, but the vein had been so much contused by the pressure of the instrument, that its coats gave way in the operation, and hæmorrhage took place to such an extent that the animal's life was despaired of. After remaining for some time in a state of syncope, it gradually rallied, but no particular œdema or stiffness of the limb was at any time observable, nor any apparent obstruction of the femoral vein. On the 28th the dog was killed, and on examination it was found that, although the coats of the vein had been considerably contused, and in a great measure divided, the morbid appearances of the vein were limited to the immediate seat of injury. That no coagulum had formed in it, and that the only evidence of phlebitis consisted in a thickening and consolidation of the vein at the seat of ligature, and some

fibrinous deposit at the point of its laceration, which was closely adherent to this part of the interior of the vein.

Upon the whole, then, we may, I think, conclude that obstruction of veins from mere vital prostration is of rare occurrence, and never, probably, the sole cause of that which is met with in phlegmasia dolens. Nor can I assent to the opinion that a coagulum thus or otherwise formed in veins acts as a foreign body, and so produces irritation and inflammation of the containing vessel. For in the cases of ligature of the iliac veins, which I have reported, it was found that the coagulum formed on the distal side of the ligature was unadherent, and the vein healthy at periods of twenty-four and forty-eight hours after the application of the ligature. Bearing in mind these facts, and that where coagula formed in veins are not speedily organised, their tendency is to undergo degeneration, softening down, and removal, we may, I think, certainly conclude that coagulation of blood in veins, consequent upon mere vital prostration, is seldom the cause of extensive or persistent venous obstruction.

But, in the next place, it has been assumed that such obstructions may arise from the admixture of morbid animal secretions with the blood, whence coagulation of this fluid immediately results, and precedes and gives rise to the phenomena of phlebitis. This doctrine has been especially advocated by Mr. Henry Lee, in some valuable papers on the pathology of phlebitis. He is led to believe, from numerous experiments, that the admixture of pus and other morbid animal secretions with the blood, hastens very remarkably its coagulation, both within and without the vessels of the body; that through this agency extensive coagulation of the blood may take place whenever these fluids are received into the veins, and that from the coagulum so formed a pellicle is separated which becomes vascular, and finally so firmly united to the circumference of the containing vessel as to be inseparable from it. I will briefly consider the grounds upon which this opinion rests before appealing to the testimony of direct experiment.

The opinion in question would appear to rest upon two series of observations, the one having reference to the effects of pus when mixed with the blood out of the body, the other to its effects when introduced into the veins. In the former case it was observed that whilst healthy blood occupied fifteen minutes in completely coagulating, that this took place in only two when pus was mixed with it. In the latter it was found that when injected into a vein, the vessel became tense during the operation, and sensibly resisted the attempts which were made to propel its contents onwards towards the heart.

Now, with reference to the first of these series of observations, I would venture to suggest that more may have been deduced from them than is strictly warranted by the facts of the case, and that an inference has been drawn from them in regard to the action of pus and other morbid secretions upon the blood, which is true also of a variety of simple and perfectly normal fluids. Incidental to this inquiry I felt it necessary to make some observations upon the rate of the coagulation of the blood, out of the body, under different circumstances, and I found that such fluids as perfectly healthy serum, or even pure water, when added to it in a certain proportion and of a given temperature, exercised a remarkable power in accelerating its coagulation, and this more particularly when the blood so treated was maintained at the temperature at which it circulates in the body. Thus, whilst I found that the blood of the rabbit began to coagulate ordinarily in two minutes and a half, and was completed in five minutes and a half, I observed that when it was received into a basin which had been placed in warm water of the temperature of 110°, and was kept at this heat, that it began to coagulate in less than one minute, and was completely coagulated in a minute and a half.—That half an ounce of blood received into a vessel placed in warm water, containing a similar quantity of healthy serum, began to coagulate in less than half a minute, and that coagulation was completed in less than one minute.—That an ounce of rabbit's blood received into a vessel containing half an ounce

of water of the temperature of 110°, began to coagulate in one minute, and that coagulation was general in two minutes. I could adduce many other observations, if it were necessary, to show that that which is true of pus in accelerating the coagulation of blood, out of the body, is true also of a variety of harmless and, as I have said, perfectly normal fluids.

But, in the second place, it was observed by Mr. Lee, that when pus was injected into the jugular vein of a healthy ass, and propelled in the course of the circulation by pressure externally, the vein became tense during the operation, and sensibly resisted the attempts which were made to propel its contents onwards towards the heart: and hence it was concluded, that the blood in the vein had become coagulated from the direct action of the pus upon it. It may, however, be observed, that the facts referred to admit of a different interpretation, whilst in such an experiment there is a source of fallacy which may not have been sufficiently allowed for. I refer more particularly to the influence of the respiratory movements in modifying the circulation through the larger veins in the immediate vicinity of the thorax, and more especially to the effect of forced or prolonged expiration in arresting the flow of blood in these vessels. Now this influence I believe to be often very considerably exerted in these operations upon the veins; for, in consequence of the struggles of the animal, and the irregularity of the respiratory movements thereby induced, the onward progress of the blood is often retarded or arrested, and thus a feeling of tension or resistance is given to the finger when placed over the injected vein, which may be extremely deceptive.

I have often observed these phenomena when injecting various fluids into the veins; and even in regard to the femoral, the feeling of resistance from this cause would sometimes be so great, as to lead to the belief that the blood must have coagulated in the vein: nevertheless, on a full inspiration taking place, the injection would again pass readily onwards towards the heart. I am therefore unwilling to believe that mere tension, resistance, or firmness

of a large vein in the neighbourhood of the thorax, during the injection of foreign matters into it, is any proof of the coagulation of the contained blood.

But admitting the fact, that the blood does actually coagulate in the vein under these circumstances, it is yet possible, as I have remarked, to give a different explanation of it; for in such a case, the pus injected must equally come in contact with the lining membrane of the vein as with the blood; and it appears to me, that its coagulation under these circumstances may as much depend upon the reaction of this membrane, thus irritated, upon the blood, as upon an action primarily set up in this fluid. We know that the lining membrane of veins very readily reacts upon the application of irritating matters; and it will hereafter be shown, that coagulation of the blood very quickly follows upon such irritation. It will also be shown, that such coagulation is at first limited to the portion of blood which is in contact with the lining membrane of the vein, and does not, in the first place, extend to the whole column. Thus in one case I found, after throwing some dilute lactic acid into the femoral vein towards the heart, that a layer of coagulum had been deposited, within half an hour, upon the lining membrane of the iliac veins and the cava, which had at first the appearance of a solid coagulum occupying the entire calibre of the vessel. On further examination, however, it proved to be but a mere cylinder of coagulated blood, the blood in contact with the lining membrane of the veins having been coagulated, whilst its interior was perfectly fluid. Such an observation appears to me to invalidate the opinion that the blood is first coagulated in the veins by the contact of irritating matters, and that the morbid changes which may take place in the vein are consequent upon and occasioned by such primary coagulation of this fluid.

Further, on appealing to the testimony of direct experiment, I was unable to verify the observations of Mr. Lee as to the formation of coagula in veins, from the introduction of puriform and other abnormal fluids into these vessels, and their direct action upon the blood irrespectively of the veins.

For in all cases in which these were used in a concentrated form, it happened that the animal almost invariably died, without, as shown on a post-mortem examination, any attempt having been made to arrest their progress onwards towards the heart by the formation of coagula in the veins; whilst in others, in which they were used in a less concentrated form, although it is true, that the vein became obstructed from the formation of coagula, yet these effects, as will hereafter be shown, were not produced by any primary action taking place in the blood, but admit of explanation in a very different manner.

In illustration of these remarks I would observe, that in several cases in which impure lactic acid, largely diluted, was injected into the veins, death rapidly ensued, without, as shown on post-mortem examination, any distinct coagula having been formed in these vessels, or any barrier attempted to its transit onwards towards the heart. In these cases, indeed, death would appear to have resulted from the impression of the acid upon the lining membrane of this organ; and this would sometimes take place so quickly, as to forbid the notion that any such barrier had been attempted. The following example may be quoted as a type of many others. The left jugular vein of a dog was exposed on the 12th June, 1852, and ʒiss of impure lactic acid diluted with ʒiss of warm water was slowly injected towards the heart. Before the injection was completed the dog became convulsed; his pupils dilated, he breathed with difficulty, and shortly afterwards died. On making a post-mortem examination, the vein down to the heart was found to be filled with a black tarry-looking matter, which adhered to the lining membrane of the vein, but in which no distinct coagula could be found. In injecting pus into the veins I have also been unsuccessful in obtaining any evidence of extensive coagulation of the blood, occasioned merely by its admixture with it. Thus in two cases in which half an ounce of pus, slightly diluted with warm water, was injected into the femoral vein towards the heart, both animals speedily died; and on examination no particular coagulation

of the blood was found to have taken place in the course of the injection, whilst purulent matter was plainly discernible in the right cavities of the heart, as shown in the following experiment.

EXPERIMENT XVIII.—The femoral vein of a dog was exposed on the 15th June, 1852, and half an ounce of pus, slightly diluted with water, was slowly injected into it towards the heart. In rather more than a minute the dog seemed distressed, the abdominal and respiratory muscles became convulsed, and respiration ceased within two or three minutes. On making a post-mortem examination shortly afterwards, the vena cava and the abdominal and thoracic veins were found generally turgid with blood. On opening the right iliac vein, a stream of dark-coloured fluid blood poured out; and on continuing the division of the veins upwards, the blood was still found to be fluid, with the exception of two or three small coagula, which were found about the middle of the cava. The right auricle contained a thin black coagulum, and small fibrinous coagula were scattered over and between the columnæ carnea of the right ventricle, which contained a minute yellowish-looking matter closely resembling the pus which had been injected.

It appears to me unnecessary to pursue this question at greater length, not only because the subject of purulent injection of veins has been very ably investigated by several pathologists, but because my object being solely to consider it as the cause of obstructive phlebitis, I think the facts brought forward sufficiently subserve this purpose, and show that it does not ordinarily arise from actions thus excited in the blood irrespectively of any agency of the veins. I do not doubt, on the one hand, the power of pus to hasten the coagulation of blood out of the body, nor, on the other, that the jugular vein may have felt hard and obstructed after two ounces of pus had been injected into it. But in regard to the former of these facts, I would observe that other agencies besides pus, and some of a strictly normal character, will

produce the same results, whilst a different explanation may be given of the latter than that which has been assigned to it. Neither do I doubt that nature endeavours to hem in noxious matters when received into the veins by the formation of coagula in these vessels; but restricting my observations to the general pathology of obstructive phlebitis, it does not appear to me to have been conclusively shown, from the facts and arguments I have quoted, that this is the usual mode in which it is produced.

Of Irritation of the Lining Membrane of Veins, the blood being excluded during the operation, and subsequently readmitted.

EXPERIMENT XIX.—The jugular vein of a dog was exposed on the 4th February, 1852; and the several veins from which it arises having been ligatured, so as to prevent any blood from entering it, an opening was made in one of its branches, and through this a solution of the nitrate of silver was passed into its interior upon lint, and applied to about an inch and a half of its lining membrane. The opening in this branch was then closed by ligature, and the ligatures taken off the other branches, so as to allow the blood to circulate again through it. The external wound was then closed by suture, and in sixteen hours the dog was killed and the vein examined.

Dissection.—The cellular sheath surrounding the vein was much inflamed, and its external coat was thickened and injected. On opening it, a firm coagulum occupied the interior of the vein to the extent to which the irritant had been applied. This coagulum was of a uniformly dark colour, and adhered at its upper and lower extremity. On removing it, the lining membrane of the vein to which the irritant had been applied was found slightly reddened and discoloured; but no distinct vessels could be seen. Small portions of fibrine were adherent to it, apparently derived from the coagulum; but no coagulable lymph had been exuded. The coats of the vein were here generally thickened

and opaque; but immediately below the irritated portion the morbid appearances almost abruptly ceased, and elsewhere the vein was perfectly healthy.

This experiment was repeated in three different instances, and with essentially the same results. In each the jugular vein was irritated by a solution of the nitrate of silver, introduced upon lint through one of its superior branches; and on subsequently readmitting the blood which had been temporarily excluded, a firm coagulum was found in that portion of the vein occupying the extent of the vessel to which the irritant had been applied.

The results of these experiments, taken in conjunction with those which have been previously related, appear to me to be not only important in themselves, but to point to the true pathology of obstructive phlebitis. We may deduce from them:

I. That irritation of the lining membrane of veins may so modify the vital properties of the blood circulating through them as to give rise to speedy coagulation of this fluid, and this to an extent commensurate with that of the irritation which may have been excited in their lining membrane.

II. That such irritation of the lining membrane may take place without any mechanical injury of the corresponding vessel; the obstructed veins in these cases having been uninjured, inasmuch as the irritant was introduced into them through an opening in one of their superior branches.

III. That coagulation of the blood in veins may take place from this cause without any primary or independent action having been set up in this fluid; for in these cases the blood was excluded from the veins both prior to and during the application of the irritant to their lining membrane. Thus, it must follow that the action of the irritant must have been exclusively upon the vein, and

that the resulting coagulation of the blood could only have arisen from some impression made upon the blood by the lining membrane thus irritated.

iv. That from such irritation of the lining membrane of veins all the phenomena of obstructive phlebitis may follow; the coagula in these cases having undergone various changes in colour and consistency, adhesions having formed between them, and the veins and the coats of these vessels having become thickened, vascular, and opaque.

v. That the morbid changes in such cases are limited to the extent to which the lining membrane of the veins may have been irritated; the blood and the veins beyond this having been found perfectly healthy in these cases.

It would thus appear that whilst phlebitis may occur in a variety of forms without giving rise to extensive obstruction of veins—whilst such obstruction is not producible by mechanical injury of these vessels, by irritation of their external coat, by actions primarily taking place in the blood, and does not arise from the effusion of lymph into their interior—it is yet readily produced by irritation of their lining membrane; that the obstructing material is not lymph, but blood; and that this is immediately coagulated in the vein by some impression made upon it by its lining membrane when thus morbidly irritated or excited.

In the experiments last related the pathological appearances of the veins were of a very marked and decided character: there was opacity, thickening, and morbid vascularity of their coats, with other changes, which together would be regarded as characteristic of phlebitis. But that extensive obstruction of veins may be excited by the passage of irritating fluids over their lining membrane without giving rise to inflammation; that, therefore, phlebitis forms no essential part primarily of the pathology of such obstruction; and that a state of mere excitation or irritation of this membrane, unattended by any appreciable alteration of

its structure, will alone suffice to give rise to it, is proved by the results of the following experiments:

EXPERIMENT XXII.—The left femoral vein of a dog was ligatured on the 23d July, 1852, and half an ounce of lactic acid, diluted with an equal quantity of water, was injected into it towards the heart. The animal was sensibly affected by the operation: he breathed heavily and irregularly, and shortly afterwards appeared to be faint and giddy and incapable of standing. He had now frequent gulpings, made efforts to vomit, and had apparently a disposition to tenesmus or diarrhœa; the breathing became more irregular, convulsions ensued, and he died in about half an hour from the period of the injection.

Dissection.—The iliac veins of the left side, from the femoral up to the cava, and a considerable extent of the cava, were found obstructed by what appeared to be a firm coagulum; and, on opening these vessels, this was found to be closely adherent to their lining membrane. After a short time, however, this apparent coagulum began to contract, and, in doing so, separated itself from the veins, exuding at the same time a serous fluid from its interior. This contraction proceeded until the coagulum, which had previously filled the entire cavity of the veins, now occupied but a small track of their interior. The coats of the veins which had been obstructed were perfectly healthy: no morbid redness, vascularity, or opacity was any where observable, and the lining membrane to which the coagulum had been adherent was perfectly smooth, white, shining, and free from any trace of inflammation.

Now, there cannot, I think, be any doubt that the blood had coagulated in these veins independently of any inflammation of the containing vessels, or that the coagulation was limited to the layer of blood immediately in contact with their lining membrane; and from these facts two inferences may be drawn: 1st, that phlebitis is not essential to the production of obstruction of veins; and 2dly, that the action of irritants thrown into the veins is not primarily or

exclusively upon the blood, and therefore, that obstruction of these vessels taking place under such circumstances does not depend upon actions independently taking place in this fluid. On the other hand, looking to the fact that it was not the entire column of blood which was coagulated in this case, but only the layer in contact with the lining membrane of the veins, it would rather appear that some reciprocal reaction had taken place between the blood and the vein, which was immediately due to irritation of the latter. The same facts are shown in the following experiment, in which, however, a longer interval was allowed to elapse between the irritation of the veins and the death of the animal:

EXPERIMENT XXIII.—The right femoral vein of a dog was ligatured on the 23d July, 1852, and ʒiij of lactic acid, diluted with ʒix of tepid water, was injected above the ligature towards the heart. In twenty hours it was found that the corresponding limb was swollen and tense, and that the dog used it apparently with difficulty and pain. He was now killed, and an examination immediately made of the parts.

Dissection.—The right femoral vein was throughout obstructed by a firm adherent coagulum, as far down as the popliteal. The right iliac veins and the cava, as far up as the renal, were also obstructed with what appeared to be a firm coagulum, and on opening them it was found that this adhered throughout to their lining membrane. By degrees, however, it was observed to contract, and in doing so to separate itself from the lining membrane of the veins to which it had been adherent, and a serous fluid was, at the same time, exuded from its interior. This contraction went on until the coagulum, which had previously filled the entire calibre of the veins, now formed a mere band within them, occupying little more than a third of their diameter. During its separation, small patches of red fibrinous matter were left upon the lining membrane of the veins; but these vessels were otherwise perfectly healthy. They were neither

thickened, vascular, nor opaque, and their lining membrane was smooth, white, and translucent, and free from any kind of inflammatory exudation.

I submit, then, that these experiments, conclusively show that obstruction of veins may occur independently of phlebitis; that phlebitis is not, therefore, an essential condition of its development, but rather a consecutive phenomenon; that such obstruction is producible by irritation or excitation of their lining membrane alone; and that such irritation may occur without any local injury of these vessels.

In the last place, I proceeded to investigate how far such irritation might be produced solely by a general vitiation of the blood; for such a presumption was rendered probable by the whole of the results of the preceding investigation. It had, for instance, been repeatedly shown that whilst wounds, injuries, &c. of veins were incapable of giving rise to extensive obstruction of these vessels in a healthy animal, otherwise in one whose blood was perfectly normal, that it yet readily followed upon irritation or excitation of their lining membrane in the absence of such causes. Reasoning, then, upon these facts, I was led to believe that such irritation of the lining membrane of veins might be produced by the presence of certain abnormal matters in the blood, circulating with it and acting upon particular veins or portions of the venous system. I accordingly proceeded to put this question to the test of direct experiment, and the result of a somewhat lengthened investigation enables me to affirm,—that not only particular veins, but considerable portions of the venous system, may become thus obstructed, in the absence of any mechanical injury of these vessels.

Experiment xxiv.—The right femoral vein of a dog was ligatured on the 16th June, 1852, and the effects of the operation were carefully noted for several days. In twenty-four hours there was some little œdema of the limb, but it was not painful or tender, and the dog moved it without

difficulty. In forty-eight hours the œdema was nearly gone. In seventy-two hours there was none perceptible, and the limb appeared to be perfectly natural. The left femoral vein was now ligatured, and above the ligature ʒiss of lactic acid diluted with ʒxss of tepid water was injected towards the heart. In twenty-four hours the corresponding extremity was greatly swollen throughout, its temperature was raised, it was painful on pressure, and the dog moved it with difficulty. In forty-eight hours its general condition was the same, the swelling was still considerable, the limb was tender on pressure, and the dog still moved it with difficulty. It was now killed, and the parts examined.

Dissection.—The left posterior extremity was generally swollen. The subcutaneous and intermuscular cellular tissue was morbidly vascular and infiltrated, and the muscles themselves were in places swollen and congested. The iliac veins of this side were obstructed with a firm coagulum, which extended somewhat into the cava, *whilst the femoral and all the principal veins of this extremity were found similarly obstructed.* On opening these vessels it was found that the external and common iliac veins were filled with a firm coagulum, which was throughout adherent to their lining membrane, and, on removing it, some red gelatinous-looking matter was left upon the interior of the veins, which for the most part could be readily brushed away, but here and there portions of it were firmly adherent. The colour of the lining membrane of these veins was somewhat redder than natural, but the membrane itself was for the most part smooth and polished. At its junction with the femoral, the coats of the external iliac vein were thickened and opaque, and it did not collapse on being cut through. The left femoral vein was filled with a dark coagulum, which was somewhat soft and grumous immediately below the ligature, but which elsewhere was of firm consistence. It was slightly adherent, and, on removing it, a little gelatinous-looking matter was left upon the lining membrane, which could be easily brushed away. This vessel was neither thickened nor

opaque, nor was any increased vascularity of its coats observable. The principal branches of the femoral vein were all filled with firm coagula, but these were very slightly, if at all, adherent to the interior of the veins. The posterior tibial vein was thickened, and its coats did not collapse on being divided; it was throughout filled with a coagulum. The right femoral vein contained a firm coagulum which was slightly adherent, and, on removing it, a little red matter was left upon its interior. This vein was neither vascular, thickened, nor opaque. A small coagulum was also found in the right popliteal vein. On making transverse sections of the different coagula found in the veins, it was observed that their exterior was much firmer than their interior. The former consisted of firmly coagulated blood, the latter of little more than a grumous semi-fluid material. Hence it seems reasonable to conclude, that coagulation had commenced at the circumference of the coagula, where the blood was in contact with the lining membrane of the veins.

Now, should it be supposed that the extensive obstruction of these veins, which followed the injection of lactic acid into the blood, was in any way due to the operative proceedings had recourse to, I would beg to observe, that no such obstruction occurred in any of the preceding experiments, in which the veins were similarly ligatured or divided; and, further, that in this the right femoral vein had been previously ligatured without any such obstruction resulting. But, to put the question beyond a doubt, I repeated precisely the same operation upon another dog, with the sole difference of injecting tepid water into the blood, instead of lactic acid, and the result proved that no obstruction whatever of the veins took place, even after a longer interval had been allowed to elapse.

Experiment xxv.—The femoral veins of a dog were both ligatured on the 28th June, 1852, and above the ligature of the right an opening was made, and ʒiss of tepid water injected towards the heart. The dog was examined regularly for several days, but no swelling was observable in

either of the posterior extremities, neither did the dog at any time appear to suffer from pain, weakness, or stiffness of either of these limbs. In seven days the animal was killed, and the veins immediately examined.

Dissection.—The pelvic veins were throughout perfectly pervious and healthy. The femorals at the point of ligature were slightly thickened and obstructed, and adhered to the surrounding parts, but elsewhere they were perfectly healthy, and no other morbid appearances were anywhere perceptible.

As, however, I was unwilling to let the question rest upon the evidence of a single observation, I repeated the same experiment in a variety of ways in different animals, and, upon the whole, with results which were affirmative of the correctness of the principle I have stated. In all, considerable obstruction of veins followed the track of the irritant in its progress onwards towards the heart, whilst in the majority there was also obstruction of the distal veins of the extremity which, consistently with the results of preceding experiments, could only have arisen from the action of the irritant after it had gone the round of the circulation. Thus, in one instance in which the jugular vein was opened, and some weak lactic acid injected, in the course of the circulation it was found that a firm coagulum occupied the whole of its interior, which extended down to the right auricle, and was in places fibrinous and decolourised. In another, in which ʒiij of lactic acid, diluted with ʒix of water, were injected from the right femoral vein towards the heart, it was found that not only had a layer of coagulum been deposited upon the lining membrane of the iliac veins and the cava, as high as the renal, but that a firm coagulum occupied also the right femoral, from its commencement to its termination in the popliteal. In a third, in which a similar quantity of dilute lactic acid was injected into the femoral vein towards the heart, it was found that not only were the iliac veins of the same side firmly obstructed, and a layer of coagulum deposited upon and adherent to the cava for some distance, but that the femoral vein of the

opposite extremity was also obstructed to a considerable extent. In a fourth, in which impure lactic acid, largely diluted, was injected into the right femoral vein towards the heart, it was found that not only were the right iliac veins and some of their branches obstructed by a firm adherent decolourised coagulum; but that the right femoral vein, down to the popliteal and several of its branches, were similarly obstructed, whilst a firm coagulum occupied also the left femoral vein down to the popliteal. In a fifth it was found where the same irritant had been injected into the femoral vein, that not only were the iliac veins of the same side and some portion of the cava obstructed with a firm adherent decolourised coagulum, but that the femoral veins contained also coagula on the distal side of the ligatures which had been applied to them.

It appears to me that the results of these experiments are essentially affirmative of the truth of the principle which I have advanced, for they show that not only may large tracks of the venous system become obstructed by the action of an irritant in its progress onwards towards the heart, but that other portions of this system may also become obstructed by its action, after it has gone the round of the circulation; and this I would submit has been shown to occur in a sufficient number of instances to justify the general conclusion, that whenever the blood is vitiated, whether from local or constitutional causes, that not only particular veins, but large portions of the venous system may, under certain circumstances, become obstructed and inflamed, and this independently of any injury of the veins themselves.

I have referred the obstruction of veins in these cases to irritation or excitation of their lining membrane, immediately induced by the presence of morbid matters in the blood, and I would now beg to explain the sense in which the term is used, and the extent to which it appears to be applicable to the phenomena in question. It has been shown, that when an irritant has been applied to the lining membrane of a vein whilst the blood is excluded, that the blood will immediately

coagulate on being again readmitted, and that consequently this coagulation must arise from some impression made upon it by the lining membrane thus irritated. It has further been shown, that this may occur without any evidence of inflammation of the vein, or apparent structural change of the vessel, and that the coagulation is limited in the first place to the portion of blood in contact with its lining membrane. Upon these data, I have assumed that the phenomena in question depend upon a disturbance of the relations which normally subsist between the blood and the lining membrane of the veins, and that this is immediately due to irritation of the latter. In this sense, then, the term is intended to signify a state of excitation of this membrane, in which the equilibrium is disturbed which naturally subsists between the blood and the membrane, and it points to that general principle of design, harmony, or adaptation in the animal economy, in virtue of which the different organs subserve their respective purposes, and respond to their appropriate stimuli. Thus we know, that pure atmospheric air gives no uneasiness to the respiratory mucous membrane, whilst impure air, or a drop of water, gives rise to violent irritation of it. That certain kinds of food are grateful to the mucous membrane of the stomach and alimentary canal, whilst others excite irritation and disorder, and so, also, in regard to the circulatory organs, we may assume that a certain quality, consistency, and vital condition of the blood, is best calculated to ensure its easy transmission through the blood-vessels. Now should these qualities, whether physical or vital, be greatly altered from their natural condition, it appears to me reasonable to conclude, that just as irritation may be set up in the former case, leading to various forms of disorder, so in regard to the latter, may irritation of the lining membrane of veins be excited without any visible change of structure; and that, in virtue of this, a disturbance may ensue in the relations which normally subsist between it and the blood. I know, at least, of no other way in which the phenomena in question can be as satisfactorily accounted for, seeing that they are not dependent upon

either inflammation of the vein or upon changes exclusively confined to the blood; and I would further observe, that this opinion is strengthened by the fact, that in cases in which the irritation of the lining membrane of the vein has been slight, extensive coagula formed in veins may soften down and be removed without leaving any trace of inflammation of the vessel behind.

Let me not, however, be supposed to affirm, that irritation of the lining membrane of veins terminates thus favorably in all cases; on the other hand, it must be distinctly understood, that whenever it has been violent or long-continued, vascular actions will sooner or later take place in the irritated vein, tending to its injury or permanent obstruction. Whenever, indeed, a layer of coagulated blood is thus deposited upon the interior of a vein, and is not speedily softened down and removed, its organisation must be expected, in the progress of which the coats of the vessel will become thickened and opaque, its vasa vasorum enlarged and injected, and the general phenomena of phlebitis developed. This is the condition in which obstructed veins have generally been met with in fatal cases of phlegmasia dolens, and hence the disposition on the part of the profession to attribute such obstructions exclusively to phlebitis.

The general conclusions, then, which appear to be deducible from the foregoing investigation, are the following:

1. That inflammation of the iliac or femoral veins will not alone give rise to all the phenomena of phlegmasia dolens.
2. That the extensive obstruction of the veins met with in this disease, is not producible in a state of health by merely local causes, such as injury or inflammation of these vessels.
3. That irritation of the lining membrane of veins independently of such local injury or inflammation may, under certain circumstances, give rise to obstruction of these vessels, and this to an extent

commensurate with that of the irritation which may have been excited within them.

4. That such irritation of the lining membrane of veins, giving rise to obstruction, and consecutively to all the phenomena of *obstructive* phlebitis, may be excited by the presence of various abnormal matters in the blood, circulating with this fluid, and acting upon particular portions of the venous system.
5. That the origin of *obstructive* phlebitis is therefore to be sought for rather in a vitiated condition of the blood, than in any local injury, inflammation, or disease of the veins.

And in arriving at these conclusions, I would venture to submit, that I have arrived at a solution of the problem which I proposed to myself at the outset of this inquiry, viz.: What are the specific causes or conditions which can simultaneously affect the sensory, the motor, the secretory, and the vascular organs of an extremity, so as to give rise to the symptoms by which phlegmasia-dolens is characterised. For in tracing obstruction and inflammation of veins to an abnormal condition of the blood, I have assigned to it a cause which can equally and simultaneously affect all these several organs; and by giving rise to irritation respectively of the nerves, the muscles, the lymphatics, the veins, and the cellular tissue of the limb, may occasion that tense, elastic, swelling, morbid sensibility, loss of motor power, affection of the lymphatics, and obstructed condition of the veins, which together constitute the pathognomonic symptoms of the disease.

It now only remains for me to consider how far the theory of the disease, deducible from the foregoing investigation, is supported by the clinical history of phlegmasia dolens. How far, in fact, it supports the opinion that the immediate cause of the disease is in the blood, and that the

affection of the veins like that of the nerves, the lymphatics, and the cellular tissue of the limb, is essentially secondary to and dependent upon an antecedent vitiation of this fluid.

The causes capable of giving rise to a morbid condition of the blood are, I need scarcely observe, of a two-fold character, the one being local the other constitutional. The former comprehend more especially unhealthy secretions, suppurations, or discharges from parts which are either inflamed, contused, or ulcerated. The latter, derangements of the primary or secondary assimilative processes, arrest or suppression of the natural excretions, redundancy in the system of effete or decomposing organic matter, epidemic or endemic influences, the products of febrile or inflammatory attacks, and diseases of the blood directly affecting its growth or development. When, then, in the history of a case we find that it has been obviously preceded and probably occasioned by such causes, we deduce a strong argument therefrom in favour of its blood origin. But, in the absence of such evidence, we may in general deduce a similar inference from another series of facts, viz., from the existence of various lesions in different organs of the body at the same time. This is a circumstance which has been constantly known to follow upon the artificial vitiation of the blood, and is one which is common, if not peculiar, to blood diseases. I propose, therefore, in carrying out this inquiry, to ascertain how far the histories of cases of phlegmasia dolens furnish sufficient evidence on these points to authenticate the theory of the disease which has been deduced from experimental investigation.

To determine this question I have carefully analysed and tabulated the principal facts of 100 cases of the disease, as reported in special treatises on the subject, or recorded in the periodical literature of this and other countries. These tables contain the particulars of sixty cases which occurred after or in connection with child-bearing, and of forty which occurred independently of this function,—the former comprise twenty deaths and forty recoveries; the latter twenty deaths and twenty recoveries. In arranging them I have

placed, under separate heads, their respective antecedents and probable causes, any coexisting affections, and the post-mortem appearances met with in fatal cases. I have also noted such general circumstances in their history as are calculated to throw light upon the question under consideration, of which the following may be regarded as the more important of the

I. PUERPERAL CASES.

Labour subsequently to which the attack occurred.—Of 35 cases in which this is noted—in 8, it occurred after the first; in 8, after the second; in 3, after the third; in 5, after the fourth; in 6, after the fifth; in 1, after the sixth; in 1, after the seventh; in 1, after the tenth; in 1, after the eleventh; and in 1, after the twelfth. We thus observe that the predisposition to the disease is considerably greater after the first and second than after any subsequent labour; and, without venturing to draw any positive inference from this fact, we may, I think, reasonably connect it with the more protracted parturient efforts, the greater injury of the soft parts, and the more considerable reaction which must occur in these than subsequent labours. Circumstances which, by inducing an inordinate disintegration of tissue, must manifestly tend to affect the general condition of the blood.

Character of Labour.—Of the 60 cases, this is noted in 38, and of these it is stated that in 22, it was natural; in 10, severe; in 1, preternatural; in 2, difficult and accompanied with instrumental injury; in 2, tedious; and in 1, protracted with retained placenta. If, then, we assume that it was natural in the remaining twenty-two, of which no mention is made, it will follow that in little more than a fourth only was it otherwise, and with a full knowledge of this fact, we cannot connect the origin of the disease with any peculiarity in the character of the labour, or with any local or mechanical injury of the uterus. Not that it is intended to affirm that mechanical injury of the uterus does not predispose to the attack, but rather that it is not an essential cause; one, indeed, of many others equally tending

to its occurrence, and all probably operating by giving rise to an abnormal condition of the blood.

Date of attack after Labour.—In 1, the attack began the day after delivery; in 4, on the second day; in 1, on the third; in 3, on the fifth; in 1, on the sixth; in 3, on the seventh; in 2, on the ninth; in 6, on the tenth; in 1, on the eleventh; in 4, on the twelfth; in 5, on the thirteenth; in 5, on the fourteenth; in 3, on the fifteenth; in 1, on the seventeenth; in 6, on the twenty-first; in 2, on the twenty-second; in 2, on the twenty-third; in 1, on the twenty-seventh; in 5, on the thirtieth; in 1, on the forty-first; and in 3 the date is not given. Now, on looking to these several dates, the first general inference which is deducible is, that there is no particular period after labour which is peculiar to the attack. But, bearing in mind that out of 57 cases in 47 it began within twenty-one days after delivery, we may deduce an argument therefrom in favour of its blood origin, for it is within that period that those great puerperal actions are inaugurated which powerfully tend to modify the general condition of this fluid, such, for instance, as the febrile reaction consequent upon labour, and the establishment of the milk, the modifications of the lochial secretion thereby or otherwise induced, the effects of injury or contusion of the soft parts, the reception of effete organic matter into the blood consequent upon febrile action, and the involution or disintegration of the uterus. These and other circumstances peculiar to the puerperal period, tend in an especial manner to modify its condition, and as such to predispose to various local diseases.

Extremity affected.—This is specified in 58 out of the 60 cases; and of these, in 1, the left arm was attacked, and in another, both lower extremities were concurrently affected. Of the remaining 56 the attack commenced in and was limited to the left lower extremity in 23; and commenced in the left and subsequently attacked the right in 10; whilst in 16 it began in and was limited to the right; and in 7 commenced in the right and subsequently attacked the left. Thus it would appear of the 56 cases that the affection

began in the left lower extremity in 33, and in the right in 23, making the frequency of the attack in the left, as compared with the right, in the proportion of about 3 to 2, whilst in rather less than a third of the whole number the opposite limb became subsequently affected.

The greater liability of the left lower extremity to the disease, as compared with the right, has been noticed by various authors, but I am not aware that any satisfactory explanation has been given of the fact. It is, however, worthy of remark that the relative frequency of the attack in these cases corresponds very nearly with that of the attachment of the placenta to the left side of the uterus as compared with the right, as given by Dr. Carriere, of Strasburg, in his Thesis on Obstetric Auscultation; for he states that of 66 cases in which the placental sound was noted, that it was heard on the left side of the uterus in 38, and on the right in 28. I do not, however, lay any great stress upon this fact in the present state of the inquiry, because the cases I have tabulated, as well as those noted by Dr. Carriere, are too limited in number to justify any positive conclusion on this point. But should it hereafter appear that the liability of the left lower extremity to the attack, as compared with the right, corresponds with the relative frequency of the attachment of the placenta to the left side of the uterus, as compared with the right, it would furnish an argument in favour of the origin of the disease from vitiation of the blood. For it is where the placenta has been attached that the uterine wound will be left on its removal, and it is from this that unhealthy secretions will be poured forth in cases of local injury, inflammation, or constitutional disorder, whilst here, also, these secretions will be most favorably placed for reabsorption, by being in contact with the orifices of the uterine veins which had been torn across by the removal of the placenta. Thus the veins of the corresponding side would be especially liable to irritation and obstruction, for it has been clearly shown that when the blood is vitiated by the artificial introduction of foreign matters into it, the effects of such vitiation are principally manifested

in the veins and organs in the immediate seat of their insertion.

The frequent disposition to metastasis, or a subsequent affection of the opposite limb, observed in these cases, is worthy of remark, and would appear to be only explicable on the ground that the proximate cause of the disease is in the blood. Such disposition being almost peculiar to blood diseases.

Antecedents and probable Causes.—In 33 of the 60 cases, the attack followed upon some form of puerperal fever; in 13, upon exposure to cold, and consequent arrest of the cutaneous excretion; in 3, upon dietetic errors, and consequent derangement of the assimilative functions; in 3, upon the operation of epidemic influences; 4, occurred in the progress of pulmonary consumption, or some other constitutional disease; 2, followed quickly upon severe or protracted labour; whilst in 3, no specific information is given on this point. Thus it would appear, that in nearly all these cases the attack had been preceded, and was probably occasioned by some circumstance capable of vitiating the blood, such as fever of various kinds, suppression of the natural excretions, epidemic influences, derangement of the assimilative processes, or some constitutional disease. To appreciate more fully, however, the influence of these causes in giving rise to the attack, it will be necessary to indicate the particular sequence of morbid actions by which it was preceded in each case.

Thirty-three Cases followed upon some form of Puerperal Fever.—The nature of the fever does not appear to have been specified in all cases, nor is it necessary for the purpose of this inquiry to investigate very minutely this point. It may be sufficient to observe, that the fact alone is important, as showing that phlegmasia dolens is in a large proportion of cases a consequence of puerperal fever; and without entering upon a discussion of its nature, how far, for instance, it originates in a vitiation of the fluids or a primary inflammation of the solids, we may assume that with its accession and development a vitiation of the fluids must ensue. I will therefore address myself more parti-

cularly to a consideration of the several circumstances which may originate phlegmasia dolens in connection with puerperal fevers.

Of the 33 cases referred to, it would appear that in 14 the fever had been preceded by a severe, tedious, or preternatural labour, or some circumstance calculated to give rise to inflammation of the uterus, or to unhealthy uterine discharges, which received into the circulation, would tend to contaminate the blood; whilst in the remainder, the labour was either natural, or not such as to have required any particular mention. We may thus recognise a distinction in these cases, between those preceded by, and those irrespective of uterine injury; and believing that they are respectively typical of the influence of local and constitutional causes in vitiating the blood, and so giving rise to the phenomena of the disease, I will treat of them separately under these heads.

The 14 cases of puerperal fever which followed upon some kind of uterine injury or lesion, present respectively the following sequence of morbid actions:

1.[1] Severe labour with hæmorrhage; sixth day, fever and pleurisy; fifteenth day, phlegmasia dolens.

7. Protracted labour and retained placenta, followed by puerperal fever, which continued till the third week, when phlegmasia dolens began.

13. Difficult labour, having the lochia much disturbed. Date of attack not stated.

18. Difficult labour, and severe instrumental injury; fourth day, puerperal fever; fifth day, exposure to cold; sixth day, phlegmasia dolens.

22. Bad health before confinement, difficult labour; sixth day, peritoneal fever; fifteenth day, phlegmasia dolens.

23. Difficult labour; third day, puerperal fever; fifth day, phlegmasia dolens.

[1] These numbers refer to the Table of References appended to this Paper, the analysis of the 100 cases having been found too lengthened for publication in the Transactions.

25. Legs swelled before labour, labour difficult; fifth day, fever; ninth, pleurisy; twenty-seventh, phlegmasia dolens.
29. Lingering and frightfully painful labour; third day, metritis; seventeenth day, exposure to cold, phlegmasia dolens evening of same day.
30. Tedious labour; metritis a few days afterwards, which remitted, but returned on the twelfth day; thirteenth, phlegmasia dolens.
34. Laceration? of vagina during labour, mercurial frictions to ptyalism; fourteenth day, phlegmasia dolens.
39. Tedious labour, lasting three days, twin birth; tenth day, peritoneal fever; fifteenth, suppression of the milk, followed by phlegmasia dolens.
43. Very hard labour, child dead and putrid, flooded twice before delivery, frequent rigors; fourth week, phlegmasia dolens.
41. Labour preternatural, child still-born; tenth day, peritoneal symptoms; fifteenth day, fever and diarrhœa; twenty-first, phlegmasia dolens.
49. Difficult labour; uterine symptoms a few days afterwards; ninth day, phlegmasia dolens.

The phenomena of these cases, following upon injury or lesion of the uterus, may, as I have remarked, be regarded as typical of those which result from various local diseases calculated to contaminate the blood, and as such are deserving of particular attention.

The general results which have been found to follow the artificial vitiation of the blood, show that two series of effects may be thereby produced, which are essentially similar to those which occur in various local diseases, attended with unhealthy secretions favorably placed for reabsorption. These respectively consist in irritation or inflammation of the part into which the vitiating matter is inserted, or of the vessel through which it is conveyed into the circulation; and secondly, in a state of febrile or constitutional disturbance, which is consequent upon its circu-

lation with the blood. But these effects, it may be remarked, will vary in different cases according to the mode in which the vitiating matter is received into the system. When directly received into a vein, as happens in some cases after venesection, and doubtless sometimes after parturition, it will generally happen that irritation or inflammation of the vein will take place concurrently with febrile or constitutional disturbance. When, however, it passes through other channels into the circulation, it may happen that constitutional disorder will first ensue, and that the veins of the part may either remain perfectly healthy or become only secondarily affected, according to those general laws which determine irritation and obstruction of veins from constitutional causes. Thus we may account for the different appearances presented by the veins in cases of an apparently similar character, the difference mainly depending upon whether the morbid secretions of the part shall have passed through them or other channels into the circulation. That the products of uterine inflammation often pass into the system through other vessels than the veins is proved by the fact, that the vessels are often found healthy in fatal cases; and that phlebitis when thus induced depends rather upon the passage of irritating fluids through them, than the extension of inflammation along their coats, is proved by the circumstance, that in some cases the morbid appearances have not been continuous, but interrupted; portions of vein having been found perfectly healthy intermediately between others which had been inflamed or obstructed. The practical deduction to be drawn from these facts is, that the causes in question may give rise to phlebitis, both in a direct and indirect manner. In the former case, by passing directly into the veins left open on the interior of the uterus by the removal of the placenta; in the latter, by passing through other channels into the circulation, and so giving rise to the phenomena of fever, out of which the affection of the veins may become secondarily developed.

In 19 cases the attack followed upon some form of

puerperal fever, which had arisen independently of difficult parturition, or of any injury or lesion of the uterine organs, as indicated in the following sequences:

2. Second day, peritonitis, with fever, which continued until tenth day, when phlegmasia dolens began.
10. Twin birth, hæmorrhage, metritis; fourth week, phlegmasia dolens, preceded by fever.
11. Obscure febrile symptoms till tenth day, when phlegmasia dolens began.
12. Third day, puerperal fever, offensive lochia, pelvic abcess; tenth day, phlegmasia dolens.
17. Fell on back before labour; fourth day, fever and pelvic symptoms, which continued until the thirteenth day, when phlegmasia dolens began.
20. Great mental distress before labour; second day, fever; third, swelling in ham; fifth, suppression of lochia and milk; eighth, abscess in calf; tenth day, phlegmasia dolens.
21. Fifth day, metritis and suppressed lochia; seventh, attack of phlegmasia dolens.
32. Febrile excitement followed labour, which continued until the twenty-first day, when phlegmasia dolens began.
33. Sixth day, peritoneal fever and suppressed lochia; ninth, phlegmasia dolens.
35. Varicose veins of left leg; fifth day, uterine symptoms and phlegmasia dolens.
36. Fifth day, puerperal fever; twenty-first day, phlegmasia dolens.
38. Attacked with fever a few hours after delivery, which continued until the tenth day, when phlegmasia dolens began.
40. Labour followed by puerperal fever; twenty-first day, phlegmasia dolens, preceded by general indisposition.
41. Third day, peritoneal fever, which continued till thirteenth, when phlegmasia dolens set in.

42. Child died during labour; third day, peritoneal fever; eighth, diarrhœa; tenth, phlegmasia dolens.
48. Fifth day, metritis and suppression of the lochia; seventh day, phlegmasia dolens.
54. Tenth day, attacked with pain in the abdomen, legs, and ankles; eleventh, phlegmasia dolens.
56. Fourth day, symptoms of metritis; seventh, rheumatism; twenty-second, phlegmasia dolens.
59. Second day, pyrexia, which had not abated on the twelfth day, when phlegmasia dolens set in.

In these cases puerperal fever would appear to have arisen in the absence of any local injury or lesion of the uterus, and may therefore be ascribed to those general circumstances connected with parturition which predispose to the disease. In nearly all, febrile derangement had preceded for some time the affection; and while from this circumstance we may learn that the latter was not the cause of the former, we may trace in it an analogy between these cases of phlegmasia dolens and those which have been described by various authors, as having occurred in the progress of fevers unconnected with the puerperal state; whilst it is probable that in each the same pathological causes may have been concerned in its production.

It may, then, be a fit question to consider, what are the peculiar circumstances which give rise to irritation and obstruction of veins in cases of fever, or in those in which the blood generally is in a morbid condition? To this question, it is difficult to give a very positive answer; but on a careful review of the several circumstances which precede the attack, we may, I think, recognise *three*, whose operation would be favorable to this result. 1. Constitutional debility. 2. A redundancy of the fibrinous element of the blood. 3. An affinity between the morbid elements in the circulation and the lining membrane of the veins.

1. *Constitutional debility.*—The general history of phlegmasia dolens, and the testimony of the highest authorities point to its frequent occurrence in depressed states of the

constitution, and to its commencement with symptoms which indicate extreme prostration. Thus the attack has often been preceded by a bad state of health, uterine hæmorrhage, or the long continuance of fever or some other malady of a debilitating character, and the operation of such causes would appear to consist, not only in rendering the excretory powers inadequate to the elimination of noxious matter from the blood, but in rendering the constitutional efforts incapable of localising the general diathesis upon any particular organ or structure. When then the organic powers are greatly enfeebled, as appears to be generally the case, in those who are attacked with phlegmasia dolens, it will happen that effete or noxious matters will accumulate in the blood in a greater proportion than they can be expelled, and these in circulating through the blood-vessels may give rise to irritation of the lining membrane of the veins in which they may be supposed to be accumulated in the greatest relative proportion.

2. *A redundancy of the fibrinous element in the blood.*—The influence of this condition of the blood in predisposing to obstruction of the veins must be obvious; and it is one which would appear to arise out of the several circumstances by which phlegmasia dolens is ordinarily preceded—viz., febrile, inflammatory, and rheumatic affections. But, further, it is shown, from the researches of Andral and Gavarret, that the fibrine of the blood is in considerable excess subsequently to parturition; that immediately after this process, and during the early period of lactation, the blood contains its largest proportion of fibrine; and that puerperal diseases are apt to occur correlatively with its amount. Let it further be added that the fibrinous element is proportionately undiminished by bleeding or hæmorrhage—that its relative proportion is sometimes higher in many states of debility and prostration than in health—and it must be obvious that the state of the constitution during the puerperal period, and subsequently to febrile and inflammatory attacks, is one which is highly favorable to the production of obstructive phlebitis.

3. *An affinity between the morbid elements in the circulation and the lining membrane of the veins.*—I venture to suggest this as a probable determining cause, from a general review of the circumstances which attend the action of poisons upon the economy—the operation of medicines and the phenomena which occur in the progress of various specific diseases. It is well known, for instance, that the action of poisons, whatever their nature or intensity, is not only definite, but limited to particular organs and structures; that the *modus operandi* of remedies is equally definite; and that the tendency of various constitutional diseases is to localise themselves upon particular parts, each observing its respective affinities. But, further, it would appear probable that these localised effects of particular remedies or poisons may in part be attributed to their being appropriated into the substance of the organs thus secondarily affected, and that to this in a great measure is due the various local affections which arise in the progress of constitutional diseases. To illustrate these views it is only necessary to refer to the general history of specific fevers and constitutional disorders; and without doing more than indicating the importance of this principle, as connected with the origin of various local diseases, we may, I think, consistently with it assume that an affinity may subsist between certain morbid elements in the blood and the lining membrane of the veins, whence irritation and obstruction of these vessels may ensue.

And if it should be asked why the veins are more especially liable to irritation and obstruction in cases of fever, or in those in which the cause of such lesions is diffused equally throughout the blood, it may be remarked that there are peculiarities in the venous as compared with the arterial system which reasonably account for such occurrence. Thus, the velocity of the circulation in the veins is much less than that in the arteries, and motion tends to retard the coagulation of the blood, and rest to hasten it. Aqueous fluids within certain limits also hasten its coagulation, and these are readily absorbed by the veins.

So also saline and various excrementitious matters hasten its coagulation, and these are directly received into the veins. And, lastly, various unhealthy secretions or discharges from parts, the dregs of the secondary assimilative processes, retained excreta, and those general causes which have been referred to, as calculated to vitiate the blood, primarily pass into the veins before being distributed to the several organs by which they are destined to be separated from the blood.

The last question to be considered in connection with this part of the inquiry is, what are the peculiar circumstances which determine irritation and obstruction of particular veins, or portions of the venous system, rather than others? Now, to this question it may be replied that the liability of the veins to take on morbid action, like that of all other organs and structures of the body, is determined by the operation of various predisposing and exciting causes. Among the former may be mentioned antecedent weakness or disease; among the latter, wounds, injuries, and surgical operations; and, as a general rule, it may be said that these veins, or portions of the venous system which may have been thus predisposed, will receive the force of any prevailing diathesis rather than others. Thus, it may be said that the utero-placental veins are predisposed to morbid action, by being torn across in the removal of the placenta; that the crural veins of parturient women are generally predisposed by the actions of pregnancy and the pressure of the gravid uterus; and that any vein, surgically or otherwise wounded or injured, becomes thereby predisposed to take on morbid action. One other circumstance only need be noted, which is the influence of cold partially applied to the body in predisposing veins to morbid action. This is true generally in regard to the nerves, muscles, and other structures of a part, and experience teaches that that which is true in regard to them is true also in regard to these vessels.

In 13 cases the attack followed upon exposure to cold;

and the particular sequence tending to the attack in these cases is given in the following summary:

5. Seventh day, exposure to cold, quickly followed by pleurisy, and this by phlegmasia dolens.
6. Three weeks after labour caught cold, upon which the attack quickly followed.
14. Fifth day, much purged during night, and exposed to cold; febrile symptoms, followed by phlegmasia dolens the evening of the same day.
24. Seventh day after labour undertook the management of the house; eleventh day, seized with rigors, followed by fever; twelfth, phlegmasia dolens.
27. Fourteenth day after labour caught cold, and phlegmasia dolens began a few hours afterwards.
29. Lingering and frightfully painful labour; third day, metritis; seventeenth, exposure to cold, phlegmasia dolens the same evening.
46. Twentieth day after labour exposed to cold, which was followed by rheumatism, and by phlegmasia dolens on the twenty-second.
53. Could not suckle infant, and probably caught cold in the attempt; eighth day, pain in left crural arch; thirteenth, phlegmasia dolens.
55. Attacked by cold, producing catarrhal and pneumonic symptoms, which were followed by phlegmasia dolens in forty-eight hours.
57. Had previously suffered from rheumatism: phlegmasia dolens followed exposure to cold.
58. Patient plethoric, and delerious in the last stage of labour; second day, phlegmasia dolens, which was caused by cold.
60. Ninth day after labour exposure to cold, followed by pain in the left side and by phlegmasia dolens.
45. Ninth day after labour attacked with pleurisy, and on the thirteenth by phlegmasia dolens.

The respective antecedents of these cases sufficiently

show that the attack of phlegmasia dolens had been immediately occasioned by exposure to cold; and in connecting them with an abnormal condition of the blood, it is only necessary to point out the general fact that, from such cause, two series of effects may be produced. The one, of a *dynamic* character, comprehends more especially a disturbance of the equilibrium of the circulation, and an intropulsion of blood, upon various internal organs. The other, of a *material* character, arises from an arrest or disturbance of the cutaneous functions, and the consequent retention of various effete or abnormal matters in the blood, which circulating with it may become localised upon particular organs or structures. Now, bearing in mind that the secretion of the skin constitutes one of the most general and perhaps constant emunctories of effete organic matter from the body, it must be obvious that an interruption to its functions must be productive of a highly morbific state of blood, and this more especially during the puerperal state, when, consequent upon febrile reaction, excessive disintegration of tissue, and other causes, a large amount of effete matter is thrown into the blood, and when accordingly the depurative functions require to be maintained in a state of unusual activity.

But we may further deduce an argument in favour of the blood origin of these cases from the general analogy which subsists between them and rheumatism. Thus in their respective origin from cold or suppressed perspiration, we may recognise a similarity of cause; in the pain, heat, swelling, and loss of motor power of the affected limb an identity of symptoms; and in the weakness and impaired condition of the limb, often left after a severe attack of either, a resemblance in their remote consequences. Between the two diseases, as affecting an extremity, there is indeed but this essential difference, that in one the principal veins of the limb are inflamed and obstructed, and in the other are pervious and healthy. Now, however, that it has been shown that the same exciting cause and the same pathological condition of the blood which gives rise to rheumatism

may also give rise to irritation and obstruction of veins, the analogy between the two is rendered more striking, and their sole difference would appear to consist in this, that in the one case the veins participate in the general irritation of the limb, and in the other are exempt. Let, for instance, a person be exposed to cold and wet, and if with rheumatism of an extremity an obstructed condition of its principal veins be concurrently produced, the symptoms of phlegmasia dolens will be developed in their most characteristic form.

Lastly, the experiments I have detailed in the course of this paper, conclusively show that lactic acid, an important constituent of the sweat, and the supposed materies morbi of rheumatism will, when thrown into the blood in certain quantities, produce marked irritation and obstruction of veins, while the presence of this acid in the blood of puerperal women, either in morbid excess, or as an abnormal agent, has been experimentally ascertained by Scherrer and Lehman. Thus, the latter informs us that Scherrer, who had paid especial attention to the occurrence of lactic acid in morbid blood, "observed during an epidemic of puerperal fever, that the blood had often an acid reaction, and as this fluid contained only free albumen and no albuminate of soda, it was clear that it must contain a free acid. Scherrer certainly did not demonstrate the actual presence of lactic acid in the blood; but as he actually separated lactic acid from the exudations which were simultaneously present, we cannot reject his conclusion that the acid reaction of the blood was due also to lactic acid. I have only thrice observed an acid reaction of the blood," says Lehman, "but under conditions similar to those described by Scherrer, viz., in a case of pyemia in a man, and in the blood of two women from six to ten weeks after delivery." ('Physiological Chemistry,' vol. i, p. 97.) I conclude, then, from a consideration of the several circumstances stated, that a vitiation of the blood from arrest or suppression of the cutaneous excretion, is a frequent and efficient cause of phlegmasia dolens.

In 3 cases the attack followed upon errors of diet, as shown in the following sequence:

16. Fifth day, fever and pleurisy; fifteenth day, improper use of wine, &c., followed by febrile reaction; thirtieth, pelvic suppuration; forty-first, phlegmasia dolens.
28. Sixth day, drank some cold ale, which was followed by fever and gastro-intestinal and cerebral symptoms; twelfth day, phlegmasia dolens.
51. Sixth day, great gastro-intestinal derangement, consequent upon eating a bulky rich meal; febrile symptoms followed, and phlegmasia dolens on twelfth day.

Having thus indicated the probable origin of the attack in these cases, I will merely suggest that the cause assigned may probably operate in a two-fold manner, in giving rise to a vitiated condition of the blood. 1. By directly furnishing a crude unassimilable matter, which passing into the circulation must necessarily give rise to an impurity of this fluid; and 2. By producing febrile disorder, and thereby an arrest or diminution of various excretory functions. Thus it has happened that the lochial, the lacteal, and the cutaneous secretions have been suddenly arrested by gastric disorder, and such arrest of these functions, coupled with the presence in the blood of the products of unhealthy digestion, may under certain circumstances prove a sufficient cause for the phenomena in question. In some cases, the error has consisted in the improper use of wine or porter; in some, in the quality, in others in the quantity of food eaten; but whatever the immediate cause, the ultimate results must be the same.

In 3 cases the attack followed upon the operation of epidemic influences, as indicated in the following sequences:

3. Twentieth day, attacked with epidemic diarrhœa; thirtieth, ditto and phlegmasia dolens.
15. Varicose veins during gestation; second day, attacked

with erysipelas, and with phlegmasia dolens a week afterwards.

19. Phlegmonous erysipelas immediately followed labour, giving rise to œdema, &c.

It is scarcely necessary to observe, in regard to all these influences, that their action is primarily upon the blood, and that the local effects observed in the progress of epidemic diseases are but secondary or tertiary phenomena. The erysipelatous poison was concerned in the causation of two out of the three cases, and as it would appear to be an important element in the pathology of many puerperal diseases, I will briefly advert to its probable relations to that under consideration.

The primary action of this poison is stated by Dr. Thomas Williams to be upon the blood, its secondary action upon the skin and cellular tissue, and its tertiary action upon the membranes of the brain and the mucous membrane of the alimentary canal; but bearing in mind that many cases of peritonitis are traceable to the erysipelatous poison, that "the infants of mothers labouring under puerperal fever often die of erysipelas, and that the assiduous attendants on puerperal fever patients are not unfrequently affected with erysipelas" (Ferguson), I think a strong case is made out in favour of the opinion that the serous membranes generally are liable to its secondary action, and if so, that the lining membrane of the veins may participate in this liability. It is certainly the case that phlegmonous erysipelas of the skin and cellular tissue of a limb often coexists with purulent inflammation of the lining membrane of its principal veins, and if in these cases it is shown that the erysipelatous poison can so affect this membrane as to give rise to purulent inflammation, it appears to me probable that the same poison, in a more mitigated form, or modified by the state of the constitution, may give rise to more mitigated consequences,—to irritation rather than to inflammation of the lining membrane of veins, or to obstructive rather than to suppurative phlebitis.

In 4 cases the attack followed upon pulmonary consumption or some other constitutional disease, viz.:

9. Phthisis before labour, in the progress of which, subsequently to parturition, the attack supervened.
37. Feeble constitution; third day after labour diarrhœa supervened, which continued until the twenty-first day, when phlegmasia dolens began.
47. Febrile symptoms after labour, bad general health, livid blotches over surface, offensive vaginal discharge; phlegmasia dolens in fourth week.
52. Bad health before labour; much gastro-intestinal derangement, which continued until the fourteenth day, when phlegmasia dolens began.

The histories of these cases fully indicate the existence of a bad state of health prior to the attack; and looking to the well-known fact that the disease has often been known to occur in connection with phthisical, gouty, and rheumatic states of the constitution, we can have no hesitation in recognising its origin from such causes, nor in connecting it in these cases with an abnormal condition of the blood.

Two cases followed upon severe and long-continued parturient action; the first, No. 21, is taken from Hull's Essay. The second, No. 50, from White's Enquiry. In the former, the attack came on in thirty-six, in the latter, twenty-four hours after delivery. As these cases are almost peculiar in regard to their time of accession, I will quote somewhat more of their respective histories than I have generally introduced in the analysis.

The subject referred to in case 21 was a young woman, aged 27, who had been rather lame during pregnancy. Her legs, especially her left, had swelled, and the veins had been enlarged and blackish during the last month. She had a hard labour, which lasted four days, and her child died the morning after delivery. In the afternoon of the succeeding day she was suddenly attacked with extreme pain in her loins and hip on the left side, and soon afterwards with pain on the outside of her left thigh, a little

below the middle. The fore part of her knee next became pained, and the pain ascended from thence to the groin and labium pudendi, and afterwards descended to the leg and foot. She experienced no rigors or other symptoms of pyrexia before the pain seized her, but she subsequently became feverish, and her lochia diminished, became thin and offensive, and soon stopped altogether. The pain was of the most excruciating character, and after many weeks had elapsed a large abscess formed in the loins where the pain had first commenced.

With regard to the origin of this attack, it appears to me that two causes may be assigned:—1st, the absorption of unhealthy secretions from the parturient surfaces, consequent upon the mechanical injury sustained by four days severe labour; and, 2dly, some incidental exposure to cold giving rise to rheumatic irritation of the parts primarily affected. The entire absence of uterine symptoms previously to the local affection, the commencement of the pain in the back and hip, and its peculiar severity, would rather favour the latter opinion, whilst the further progress of the case might have been modified by the absorption of unhealthy discharges from the uterine organs. Feeling, however, uncertain as to the origin of the attack, I do not think myself justified in pursuing the question any further.

The history of case 50 is given very briefly: the patient was aged 34, her labour was very violent, and it is stated that the child rested a long time at the brim of the pelvis. Whilst she was in a standing posture, it is further stated, that she had a strong pain, and thought she perceived something within her break, just above the right groin. She was delivered upon the knee the next pain. In twenty-four hours after she was seized with violent pain and swelling in the groin and labium pudenda of the right side, which descended to the thigh and leg. Her lameness and swelling continued till the middle of her next pregnancy, and were always much increased upon walking.

The principal difficulty in this case consists in determining the nature of the supposed rupture, and its influence

in the causation of the disease. Such a circumstance appears to be peculiar in the history of these cases; and in the absence of any exact knowledge of the injury, it is difficult to know what consequences to assign to it. It should be borne in mind, however, that the reporter of this case was advocating a theory which would be supported by the fact referred to, viz., that the proximate cause of the disease consists in a rupture of the lymphatic trunks at the brim of the pelvis. The only other circumstance noted in the history of this case is that the labour had been very violent.

It would appear, that in one or two instances the attack followed upon sudden suppression of the milk; but not knowing how far it was attributable to such cause, I have not made a separate group of these cases. The following case, however, bears so closely upon the present inquiry, that I venture to introduce it. It occurred in the practice of MM. Tessier and Recamier. "A woman during lactation, experienced after a meal, during which she had felt much oppressed by heat, a sudden and total suppression of milk. Symptoms of meningitis soon displayed themselves, for which she was brought to the Hôtel Dieu. The inflammation was subdued by active measures; but the patient was soon after attacked by fever, attended with cough, expectoration, and all the symptoms of pneumonia. This in its turn gave way to a mild peritonitis, and this again to phlegmasia dolens." ('Gazette des Hôpitaux,' Jan. 21, 1843.) Now, whether we consider the suppression of the lacteal secretion in this case, as the cause of the several maladies which ensued, or not, it is equally important, as demonstrating in their succession and variety the connection of phlegmasia dolens with some constitutional condition.

It is thus shown, with the exception of the two cases I have referred to, that in 57 out of the 60 cases tabulated, the attack of phlegmasia dolens was preceded, and probably occasioned by some cause calculated to vitiate the blood, and with regard to the remaining three, I find on looking to their concomitant affections and post-mortem appear-

ances, that in 2 there was evidence of coexisting disease in other parts of the body. Thus in case 4, there were symptoms of inflammation of the lungs or pericardium, and in case 8, there was found after death two pints of thin purulent fluid in the left pleura, extensive recent adhesions between it and the lower margin of the superior lobe of the left lung, which portion was extensively inflamed, and contained about an ounce of cream coloured pus, whilst the inferior portion of the right lung was solidified and adherent to the pleura. I venture to suggest, that the coexistence of these several lesions with the affection of the extremity points to a common cause, which can only have existed in the blood.

Having thus deduced from the general history of these cases their probable origin from a morbid condition of the blood, I will only observe, with reference to their concomitant affections, that in 45 out of the 60, or in three fourths, there was some evident local or constitutional disorder co-existing with the malady. In 14, or in about one fourth, there was some form of thoracic inflammation or disease. In 14 there were symptoms of peritoneal inflammation, or of some disease of the abdominal viscera. In 9, or in rather less than one sixth, there were cerebral symptoms; whilst in many, various local lesions coexisted with the affection of the extremity. It would be inconsistent with the limits of this paper to enter into an analytical examination of all these circumstances; and having therefore indicated them thus generally, I will proceed to a consideration of the non-puerperal cases of the disease.

II. NON-PUERPERAL CASES.

These comprehend as already stated, 40 cases, of which 20 proved fatal, and 20 recovered. About two thirds the number occurred in females, and one third in males. I will commence with an analysis of their respective antecedents, which gives the following general results. In 10, or one fourth the whole number, the attack followed upon febrile derangement. In 11, or rather more than one

fourth, it followed upon exposure to cold. In 8, or one fifth, it occurred in the progress of pulmonary consumption, or some other constitutional disease. In 7, or in about one eighth, it followed upon the operation of local causes calculated to vitiate the blood, such as malignant ulcerations, &c. In 3, it followed upon suppression of the menses; whilst in 1, the antecedent history does not point to any particular cause.

The respective antecedents in each of these cases are given in the following summary.

In 10 cases the attack followed upon some form of fever, as shown in the following instances:

83. Had suffered from fever, and been largely depleted; seventeenth day afterwards, febrile symptoms set in, and the attack began.
84. Was convalescing from fever and intestinal inflammation, and whilst greatly debilitated the attack began.
85. Fever, followed by agueish symptoms, during which the local affection was discovered.
86. Patient convalescing from fever, and whilst greatly debilitated the attack began.
62. The attack began with fever, sickness, vomiting, and pain in all the limbs.
63. Before attack began, had frequent rigors and profuse perspirations.
64. Fever, headache, and pains in the side, for which she was bled with relief, preceded the attack.
65. Got wet four days before attack, which was followed by fever, subsequently to which phlegmasia dolens began.
76. Had been suffering for some time from fever, and was much reduced and emaciated when the attack began.
87. Gastric fever, &c.; three days afterwards catamenia appeared scantily, and next day the attack began.

In 11 cases, the attack followed upon exposure to cold, as indicated in the following series:

66. Had stood for two days, at work in a ditch, above his knees in water, fever followed, and in seven days phlegmasia dolens began.
74. Exposure of left lower extremity to cold, the patient being thinly clothed.
82. The attack was attributed to exposure to cold.
89. Exposure to cold: inflammation of the bowels and free depletion preceded the attack.
92. Got wet through when fatigued, and attack followed in two days.
93. Bilious fever, when convalescent exposed to a draught of cold air, followed by rheumatism, and then by the attack.
95. Bilious derangements, exposure to cold, and rheumatism, preceded the attack.
96. General bad health and exposure to cold preceded the attack.
97. Irregular menstruation and frequent exposure to cold preceded the attack.
98. Got wet through and had rheumatism, a week afterwards the attack began.
99. Attack preceded by rheumatic fever, brought on by lying upon the grass when much fatigued.

In 8, the attack occurred in the progress of pulmonary consumption or some other constitutional disease, as shown in the following summary:

67. Had laboured under symptoms of phthisis for nine months before attack.
68. Had laboured under symptoms of phthisis for sixteen months before attack.
61. Had suffered from rheumatism and pains in various parts of the body before attack.
75. Had been for several years in a bad and delicate state of health prior to the attack.

77. Had laboured for some time under diabetes mellitus, and had evidently tubercles in the left lung when attack began.
78. Had been addicted to drinking, and had gastric fever and pneumonia previously to the attack.
80. Bad health, hectic fever, and phthisis, preceded the attack.
81. Had been ill a fortnight before the attack began.

In 7 cases, the attack followed upon the operation of local causes calculated to vitiate the blood, such as suppurating sores, malignant ulcerations, &c., as follows:

71. Malignant disease and extensive ulceration of the uterus, preceded and attended the attack.
72. Cancerous ulceration of the uterus preceded and attended the attack.
73. A sanious discharge from the vagina and extensive ulceration of the uterus preceded and attended the attack.
90. Wound over a branch of the right saphena preceded the attack.
91. Varicose veins and an ulcer above the left malleolus.
94. Operation for the removal of a uterine polypus, followed by offensive puriform discharges; phlegmasia dolens nine days afterwards.
100. Operation for lithotomy; phlegmasia dolens thirteen days afterwards.

In 3 cases, the attack followed upon sudden suppression of the catamenia, which in 2 was occasioned by exposure to cold.

69. Sudden suppression of the catamenia from immersion of the body in cold water, immediately preceded the attack.
70. Sudden suppression of the catamenia followed by violent fever preceded the attack.
80. Attack referred to the sudden suppression of the catamenia from exposure to cold and wet.

It is scarcely necessary to point out the close analogy which subsists between the antecedent circumstances of these cases and of those which followed upon parturition. In some respects, indeed, they are identical, as in the instance of fever, constitutional disease, and exposure to cold; whilst in others, although the actual antecedents are dissimilar, we may yet recognise a resemblance in their ultimate modes of action. Thus, the influence of ulcerative disease of the uterus, independently of parturition, may be likened to that of inflammation of this organ subsequently to labour, inasmuch as both furnish unhealthy secretions capable, on reabsorption, of vitiating the blood; whilst we may also recognise a similarity between the effects of suppression of the lochia and that of the catamenia.

I proceed, in the next place, to an analysis of their concomitant affections. Of these, in 7 none are noted; whilst in 1 it is stated that the health of the patient was perfectly good. Of the remaining 33 the following is a general summary:—In 6 the local affection coexisted with some form of febrile or constitutional disorder; in 8, with thoracic symptoms, together with febrile disorder; in 2, with thoracic and abdominal symptoms; in 2, with thoracic, abdominal, and pelvic symptoms; in 3, with abdominal symptoms and some kind of constitutional disorder; in 2, with pelvic symptoms; in 3, with cerebral symptoms and constitutional derangement; in 3, with malignant ulceration of the uterus; in 1, with various local affections; in 1, with muco-enteritis, bronchitis, and ophthalmia; in 1, with delirium tremens, gastric irritation, disease of the lungs, pleurisy, and anæmia; and in 1, with diabetes mellitus, phthisis, pneumonia, pleurisy, and aphthæ. I need offer no commentary upon these facts as supporting the constitutional origin of the disease.

And turning, in the last place, to the evidence afforded by pathological anatomy, I find in all the fatal cases referred to in which a post-mortem examination was made, that it revealed the existence of various lesions in different organs of the body independently of those which were met

with in the veins of the affected extremity. In a large proportion there were appearances of inflammation in one or more of the serous membranes. In some the viscera were found diseased; whilst in others evidence of a diseased condition of the blood was furnished by the presence of fibrinous concretions in vessels remote from those of the affected extremity. These, with other points important to the present investigation, and favorable to the opinion which I have expressed respecting the nature of the disease, are fully set forth in the tables presented to the Society.

I conclude then this investigation, by observing that the results of clinical experience essentially harmonise with and support those which are deducible from physiological research; that both indicate the origin of phlegmasia dolens from a primary vitiation of the blood; and that, although the particular causes may vary in different instances, they all tend to the production of an abnormal condition of this fluid, out of which all the known phenomena of the disease may be deduced. Thus, in one series of cases we observe the attack to follow upon the long continuance of febrile action and the unhealthy condition of the blood thereby induced; in another, upon the operation of local causes calculated to vitiate this fluid, such as inflammatory, suppurative, and ulcerative diseases; in a third, upon arrest or suppression of the natural excretions, such as those of the skin, the lochia, or the catamenia; in a fourth, upon the operation of epidemic influences or poisons; in a fifth, upon derangement of the assimilative processes; in a sixth, upon constitutional disease of various kinds; and doubtless the series might be extended to many others equally tending to the causation of a vitiated condition of the circulating fluid.

Lastly, in submitting this inquiry to the profession, I venture to believe that it will not be altogether unimportant in its application to practice, and that a knowledge of the constitutional or blood origin of phlegmasia dolens will give greater effect and precision to our treatment of the disease. It would be inconsistent with the limits of this paper to enter at length upon this subject, nor would it be becoming

in me to point out to the members of this Society how the indications deducible from the foregoing inquiry can be best fulfilled. I may, however, be permitted to observe, that, without neglecting any of those measures which are required by the character of the local affection, that the treatment throughout should be of a general and eliminating character; that at the commencement of the attack emetics may be usefully given, not only with the view of equalising the circulation, but for the purpose of acting promptly and powerfully upon the secretions; that the actions of the liver and alimentary canal should be moderately sustained by mercurials and aperients; that active and continued diaphoresis should be promoted by such remedies as the acetate of ammonia, antimonials, opium, and colchicum, together with the external application of warmth and moisture, until the acute stage of the disease shall have subsided; that alkaline drinks or the nitrate of potass may be advantageously given both during the early and advanced stages of the disease for the purpose of correcting any acrimony of the fluids, and of dissolving, and thus aiding, the removal of the fibrinous coagula by which the veins are obstructed. These, with other measures calculated to subserve particular indications, as well as those of a local character which experience has sanctioned, I need not further enlarge upon, inasmuch as their various modifications will readily suggest themselves to the practitioner, by whom alone their application to particular cases can be properly estimated and determined.

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ON SOME POINTS OF

THE PATHOLOGY OF YELLOW FEVER.

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COMMUNICATED BY

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I feel that some apology is due for introducing a subject that has given rise to such fierce and unbecoming controversy as yellow fever; but an intention to avoid what have really become party questions, and a hope of being able to supply some facts of practical value, have induced me to offer for consideration and discussion the following observations, which, I believe, will be found to describe some pathological considerations of considerable importance that, as far as I can ascertain, have hitherto been overlooked, or referred to in a manner from which it is manifest their value was not recognised.

Of all diseases, yellow fever is perhaps the most insidious. It frequently happens that patients who have been pronounced convalescent, or free from danger, by practitioners of great experience and observation, have suddenly and unexpectedly expired, without leaving any post-mortem appearances by which the physician could explain the event,

or derive indications by which his prognosis might, in future cases, be more correctly formed; *e. g.*, a patient has passed through one or two violent accessions of fever, which have subsided, and return no more. He expresses himself much relieved, or perhaps feeling quite well; the tongue is clean; the pulse 70, round and soft; the skin cool, intelligence perfect, and the secretion of urine apparently natural. Notwithstanding all these favorable indications he suddenly expires.

Cases, such as the one I have related by way of illustration, occasionally occurring in my own practice, perplexed me considerably. Theorising upon the subject with the view of discovering some plausible explanation of the phenomena in question, it appeared to me unlikely that any diseased condition of the solids (fatty heart excepted, being inapplicable to the present case,) could so suddenly produce death without having previously afforded some indication of its existence, and therefore that the cause of death would be found in the blood or its circulation. Bearing in mind the extreme congestion of the capillaries of the skin (usually existing in this class of cases), I imagined that the capillary system of all the organs and tissues of the body was in a similar state, and that the congestion suddenly increasing (from many causes which may be readily supposed) was in itself sufficient to bring the circulation to a stop, and thus to cause sudden death.

While in this state of uncertainty, I accepted the appointment of physician to an hospital, where I became the colleague of Dr. Lacaille, a French physician. In conversation he stated to me his conviction that, in the cases referred to, death was caused by the formation of fibrinous clots either in the cavities of the heart or in the large vessels leading to them. He had records of two hundred post-mortem examinations, made upon individuals who had died of yellow fever, and in no instance, where death had suddenly occurred, was the clot absent. The observation, however, derives its greatest importance from the fact that the clot is to be found either in the heart or large veins, in every case of

death by yellow fever, where depletion has not been largely practised.

I became attached to the hospital referred to at Rio de Janeiro in February, 1852, and between that month and the end of May, I made upwards of fifty post-mortem examinations of individuals who had died of yellow fever, and whose cases I had observed during life. A clot was found in every instance, with the exception of those cases which had been largely blooded, and even these did not always form an exception.

It will, I have no doubt, be urged that the state I have described is a dying or post-mortem one. A sufficient refutation of this opinion is the fact that it can be diagnosed during life, two, three, or four days before death, and as early, sometimes, as the close of the second day of disease, while the system is still vigorous, the fever acute, and the functions of life, though greatly disturbed, are performed in full activity.

Sometimes no symptoms exist to denote the mischief that is going on; but generally there is great distress or fatigue in the breathing, and an indescribable anxiety is felt at the præcordia or under the sternum. Upon auscultation, the heart, in an early stage, is generally found to be beating violently, but communicates very little impulse to the ribs, suggesting the idea of a small distant heart impetuously acting. The pulse, though at first not much affected, soon becomes contracted, small, and soft, affording a remarkable contrast to the forcibly acting heart; occasionally, however, it retains its volume, notwithstanding the existence of the clot, a peculiarity depending probably upon the position of the latter. The sounds of the heart's action are either very much subdued, or one or both are entirely lost. When the formation of the clot takes place very rapidly, a species of rumbling or churning sound accompanies the disappearing of the natural sounds. Both sounds become less distinct as the disease progresses, but I cannot assert that either one is always and regularly lost before the other. In some instances the first, and in others the second, seemed to be that which

was first affected. It is generally the second sound which is first and most seriously affected. The irregularity probably depends upon the different positions occupied by the clots. When found in the heart, the clot always exists in the right ventricle, or ventricle and auricle, but is usually accompanied by another of smaller size in the left. There may also be clots in the venæ cavæ, and sometimes, but less commonly, in these alone.

That the clot directly gives rise to the formation of a rumbling or churning sound is extremely probable; but I imagine that the feebleness or loss of the natural sounds is, in some measure, owing to the obstruction the circulation experiences in the right ventricle, whereby but little blood passes through the lungs to the left side. Hence, also, the pain, or rather the distressing and indescribable anxiety felt at the præcordia or under the sternum, and the catching or sighing respiration.

These surmises require further observations to confirm.

The clot sometimes almost entirely fills up the cavity of the ventricle, or of the vessels in which it is found, and would, if existing during life, be quite sufficient to obstruct the circulation to an extent that would prove suddenly fatal, and that would satisfactorily account for the loss of the heart's sound, both by a want of circulating fluid and by the clot mechanically interfering with the movements of the heart's valves.

The clot is generally more or less round, of a clear bright amber colour, of a gelatinous appearance, but, upon closer examination, is found to be much tougher, and evidently fibrinous. By preservation in spirits it becomes opaque, of a dirty grey colour, and shrinks somewhat; sometimes the surface or the extremities of the clot have small portions of coagulated blood adhering to them. For other peculiarities I refer to the preparation now exhibited. It was taken within one hour of the patient's death,—a lapse of time that would hardly suffice, as a post-mortem phenomenon, for the formation of such large and perfectly bloodless clots, found both in the ventricles and in the large

blood-vessels. Upon examining the right ventricle of the preparation exhibited, it will be seen that the fibrinous mass interlaces itself with the carneæ columnæ and chordæ tendineæ, tying them, and evidently interfering considerably with their movements, and perhaps so far affecting the action of the valves as to co-operate with other causes in diminishing the intensity of the natural sounds. I have not discovered any abnormal bruit in these cases, further than the churning sound already referred to. The clots in the ventricles are so much smaller than those which are frequently met with, that a very imperfect idea will be formed of the extent to which they are capable of obstructing the circulation, always supposing that they existed during life. The chief value of the observations just made depends upon substantiating this fact. The following considerations convince me of its truth:

1st. I have found them as bloodless and as perfect within an hour of death as at any subsequent period at which an examination may have been made.

2d. I have diagnosed their existence several days before death in nearly a hundred instances, all of which, as I predicted, proved fatal. In upwards of fifty of these cases I verified, by post-mortem examination, the absolute presence of the clots; the remaining cases were not examined. I admit that the evidence is not conclusive; but its value is considerably enhanced by the fact that I have never diagnosed the presence of a clot in a case that recovered; so that, be the explanation what it may, an examination of the condition of the heart has given me a clearness and certainty of prognosis, in a most insidious disease, which I did not before possess. It is hardly possible that the result should be purely accidental. During the three months that my attention was particularly directed to the state of the heart in yellow fever, I attended upwards of three hundred cases, and the result verified in every instance a diagnosis, founded upon an examination of the condition of the circulatory apparatus.

It occasionally happened that the signs which I have

mentioned as indicating the presence of clots in the heart were so obscure that no diagnosis could be confidently formed; and in these cases it was necessary to suspend the judgment until the disease was further developed, or to form a diagnosis from other sources of evidence.

It is true that the clots do not possess such a clearly laminated appearance as would indicate their slow formation; and it thus becomes a question whether it be compatible with our ideas of the formation of clots from diseased blood, that those described should take place during life; and, secondly, whether these ideas are sufficiently precise and irrefragable to outweigh the evidence which I can adduce in support of the opposite view.

3d. I have found fibrinous clots in other organs, as will be presently mentioned, that must necessarily have been formed during life.

4th. The supposition that a clot exists in the heart, will frequently explain events that are otherwise inexplicable.

The only counter-argument that appears to me of much weight, is the fact of the clots not possessing distinct laminæ, from which it may be inferred that their formation was not progressive or during life. It should, however, be remembered, that I am describing a change which takes place in a highly-diseased condition of the blood; and I feel pretty sure that I have seen fibrinous exudations, the result of inflammation in broken-down strumous constitutions, with no better traces of laminæ than the clots now presented for examination.

It has been mentioned that clots have been found in other organs besides the heart and large blood-vessels. The allusion related to fibrinous exudations, or distinct clots, which upon two occasions I found in the ureters of individuals who died of yellow fever. Although I can only mention two instances in which I observed the occurrence, I have no doubt that it frequently happens in a smaller degree. In those referred to, the ureters were so completely blocked up by the masses, that it was sufficient to obstruct in toto the flow of urine. The fibrinous exudations

more generally occur in the tubuli uriniferi; at least, I think as much may be justly inferred from what I am about to relate.

Suppression of urine is not uncommon in severe cases of yellow fever, and it is almost invariably fatal. Out of many hundreds, I can recollect but one instance (*in which it was ascertained by the catheter*, that suppression of urine existed and continued for twenty-four hours,) that the patient recovered. This man was bled largely three times in twenty-four hours, and made a good recovery. Suppression occurred early in the disease, and was treated by active venesection.

I very much regret that the investigations which I made upon the diseased conditions of the kidneys and of the urine in yellow fever do not possess that precision which the subject admits of. I am no analytical chemist, and practically unacquainted with the use of the microscope. I have been obliged to dispense with the assistance to be derived from these two branches of science, very important, I confess, in the present inquiry. I must, therefore, content myself with relating faithfully the changes and abnormal appearances observed by the unaided senses; and I hope that although my observations may be meager, compared with the richness of the subject, they will suffice to show the importance of conditions hitherto, I believe, unknown, and also to impart, as far as the observations extend, some definite and practical information upon the subject discussed.

It will not be expected that in a disease of such varying intensity as yellow fever, there should be a perfect uniformity of symptoms. During an epidemic, many individuals pass through a mild species of fever, that after a single paroxysm which has not confined the patient to bed, returns no more. Between this and the most intense form every variation of degree is met with. It is important, however, to remember that a fever, which during the first two or three days appeared to be of the mildest possible kind, occasionally assumes suddenly the worst features of the disease, and runs a rapidly fatal course.

It rarely happens that a patient suffering from yellow fever does not pass albuminous urine. In the slightest cases, the albumen may be, and generally is, so small in quantity, as to be scarcely discernible; but in every instance where the fever is well marked, the presence of albumen may be readily detected. In some instances I have known the urine, when heated, to become an entire solid mass.

In cases which at the beginning appeared to be mild, but afterwards proved fatal, I have derived considerable assistance in forming my prognosis by an examination of the urine, the quantity of albumen being altogether disproportionate to the apparent severity of the attack.

When the urine is scanty, the fluid passed through the kidneys appears like turbid serum, with small particles, apparently of lymph, floating through it, the whole being very coagulable. It has neither urinous nor aromatic odour, and I presume that, if analysed, it would be found deficient in urinous principles, and probably without any urea. This condition of urine is, I believe, as certainly fatal as total suppression, appearing more like an exudation of serum than a secretion of urine. If it be scanty and coagulable, but of a urinous character, the patient may escape.

In cases of turbid serous-like urine, and also of a scanty or suppressed urine, I cannot but think that the tubuli uriniferi are partially or entirely blocked up by exudation matter. The fluid which escapes evidently contains exudation matter in abundance; and this would account most satisfactorily for the scanty or suppressed secretion. The kidneys when examined are found congested, and the papillæ yield upon pressure a glutinous tenacious exudation, sufficient, I believe, to interfere mechanically with the escape of urine. Whatever may be the explanation, this remarkable condition of the urine is very important in a disease where suppression is so common that, in the Rio de Janeiro epidemics, about eighty out of every hundred fatal cases are attended by this symptom.

Although all may not concur in the conclusions I have

arrived at, especially that referring to the formation of clots in the cavities of the heart during life, it will receive general assent, that the points discussed possess considerable value, as they at least afford evidence of the diseased state of the blood, and of the great disposition in yellow fever to the throwing out of albuminous or fibrinous matters, owing probably to their excess.

Blood drawn in yellow fever is generally very dark coloured, and sometimes almost black; but when the circulation is more than usually rapid, it may retain its bright red colour. It may be considered a very bad indication when the colour of the blood does not brighten under the effects of bleeding. The appearances of the blood drawn vary considerably; sometimes it remains very black and carbonaceous looking, with little separation of the serum; it may brighten by exposure to the air, and form a tolerably healthy-looking clot; it often throws out a thick buffy coat, the fibrin of which is seldom firm, and is sometimes so soft as to resemble a thick mass of size on the top of the clot. I have in such cases seen the fibrinous matter nearly as thick as the rest of the clot, and so gelatinous in appearance as to afford some explanation of the reason why the clots formed in the heart and large blood-vessels are not more distinctly laminated.

Without presuming to offer a theory to explain fully the nature of a disease which even now finds advocates, who complacently solve the question by a reference to contagion, I may suggest the necessity of examining the condition of the blood, in order to discover the first evidence of disease.

ON THE TREATMENT

OF

OBSTINATE STRICTURES OF THE URETHRA

BY

EXTERNAL INCISION UPON A GROOVED DIRECTOR.

BY

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My sincere and firm persuasion being that I have devised an easy, safe, and effectual method of affording relief from one of the most obstinate and distressing infirmities to which the human body is liable, I naturally am desirous that the benefit thus placed within reach, should be extended to those who require such assistance. But as the reception of this proposal has been greatly impeded by incorrect statements, I labour under the impression, that although the truth may, and no doubt ultimately will prevail, a long time must elapse before the effect of what has been said and written can be effaced through the ordinary course of experience. In these circumstances it has seemed to me, that the best plan was to come frankly before this body, which represents the medical profession in London, to explain the plan of treatment which I have endeavoured to introduce, to state the facts upon which I am willing that its credit should rest, and to supply any farther information that may be required for enabling every member of the Society to form an independent opinion upon the subject.

In proceeding to attempt the accomplishment of this

undertaking, I trust that it will not be necessary for me to detain the Society with elementary observations on Stricture of the Urethra, my object being to direct their attention to certain forms of the disease which resist the hitherto established means of treatment, and seem to require some other remedy. In one of these forms the stricture is distinguished by an extreme degree of irritability, occasioning more than usual distress to the patient, and resenting, by violent local and constitutional disturbance, all attempts to effect dilatation; in another, the stricture, when dilated, speedily contracts again, so as to renew the symptoms attending it; and in a third, the stricture, though it may remain dilated sufficiently to permit the introduction of full-sized instruments, still continues to render the urinary evacuation painful, difficult, and uncertain.

For the remedy of these three forms of stricture, the established means of treatment, which may be all referred to dilatation, caustic, and internal incision, have too frequently proved unavailing beyond temporary palliation, or have aggravated the evil. I shall not attempt to calculate how many lives are thus rendered miserable, or how many have been cut short through vain struggles between the disease and attempts to remedy it, but may simply notice the Will of the late Mons. d'Argenteuil, who bequeathed funds sufficient for bestowing, at stated periods, a prize of nearly five hundred pounds in value, for the greatest practical improvement in the treatment of stricture. It would be difficult to express more emphatically the misery endured from this source, or the deficiency experienced in the resources of surgery for its relief.

The simple bougie is quite sufficient for the relief of stricture as it presents itself in the ordinary course of practice. But for the effectual remedy of those, which assume one or other of the peculiar forms to which the attention of the Society has been directed, I am prepared to maintain that a free division of the contracted part of the urethra is essentially required. For this purpose I introduce a grooved

director; and I believe that there is no stricture which through time and care may not be made to admit a proper instrument. The patient being then placed upon his back at the edge of a bed, and the knees held up, an incision about an inch and a half in length is made exactly in the raphe of the perineum, sufficiently deep to allow the director to be felt, and to permit the knife to be inserted into its groove, when the whole thickened, contracted, and indurated texture is freely divided to the extent of an inch, or two, or more if necessary, a number eight silver catheter being afterwards retained in the bladder for at least two, and not more than three days.

The procedure which I have now described is extremely simple, and may be easily accomplished, without any hurry, in less time than has been required for explaining its different steps. But, nevertheless, it is by no means easy, in the sense of being so, to every one who chooses to undertake it; on the contrary, there is no operation in surgery which more than this demands the most exact precision of performance, whether the attainment of its object, or its immediate effects, be held in regard. Unless the incision of the urethra is at the proper place, and of sufficient extent, no permanent benefit can result, and great embarrassment with its attendant evils may be experienced in introducing the catheter; while, if the knife is not confined to the middle line of the perineum, or is allowed to slip out of the groove, there will be the greatest risk of hæmorrhage and extravasation of urine. The Parisian Imperial Academy of Medicine, in awarding the Argenteuil prize in September last to Mons. Réybard, for extending the plan of internal incision, said, "that whatever be the form of the stricture, the incision ought always to be directed laterally, so as to avoid the artery of the bulb placed below." But in a reclamation relative to the disparaging terms in which the Academy thought proper to mention my method of treatment, I took the liberty of suggesting to that distinguished body, that, with regard to the position of the artery of the bulb, they had promulgated an anatomical error of the gravest

character and most dangerous tendency in the practice of surgery; since the vessel lies at the side of the canal, and can be avoided with certainty only when an incision is made exactly in the middle line. The exactness of incision being thus of such paramount importance, I have learned with extreme surprise that a surgeon has performed the operation professedly according to my principles, but without any grooved conductor, the only guide being a small silver catheter, upon which I do not hesitate to affirm it is absolutely impossible to make a straight continuous incision through the thickened textures of a stricture. I have read with hardly less surprise, that after the operation the catheter, instead of being retained only two, or at most three days, has been kept in the bladder six weeks, notwithstanding the long-established and well-known fact, that the presence of such an instrument, so far from promoting, greatly impedes, if it does not altogether prevent, the closure of a fistulous opening into the urethra. With feelings of, if possible, still greater astonishment, I have heard of operations being performed, ostensibly in accordance with the principles which I have endeavoured to establish, but in reality without any instrument at all being introduced through the stricture.

For the results of such proceedings, it is quite obvious that the operation which I have proposed cannot be held responsible. The only sources of danger alleged to exist are hæmorrhage and extravasation of urine. But if the knife is prevented from deviating beyond the middle line, there can be no bleeding except from the smallest twigs of the superficial perineal artery and the cells of the corpus spongiosum. There is, accordingly, at the time of the operation, seldom more than a teaspoonful of blood, and afterwards an oozing which hardly exceeds the extent of one or two ounces. If the patient is very full blooded, the quantity discharged in the latter way may possibly extend even to the amount of a teacupful, and so far as I have been able to observe, rather with a salutary effect than otherwise. But if, either to prevent soiling of the bed-clothes, or to protect

a nervous patient from unnecessary alarm, even this small amount of bleeding be deemed objectionable, it admits of being easily restrained, by placing a piece of folded lint between the edges of the wound, and applying the slightest degree of pressure for a few hours, or rather minutes, since coagulation soon takes place and prevents farther escape.

In regard to extravasation of urine, there can be no doubt that the circumstances most favorable to its production are openings through the deep fascia of the perineum, together with obstructions in the anterior part of the urethra; and hence the dangers of the operation for strictures deemed impermeable, which consists of deep incisions into the perineum, with uncertain and imperfect division of the contracted part of the canal. But according to my proposal, the only fascia concerned is that which lies immediately under the integuments; the knife is guided with unerring certainty through the whole extent of stricture, and a full-sized catheter is retained in the bladder until the cut surface is sealed up by effusion of lymph, so that extravasation of urine is impossible, even less likely to happen than hæmorrhage. It was these considerations which originally led me to regard the operation as free from danger; and having now performed it upwards of seventy times without any fatal, or even alarming consequences, I hope it will be allowed that my anticipations have not been unduly sanguine.

In illustration of what has been said, I now propose to mention some cases of stricture which have resisted the ordinary means of treatment, and yielded to external incision. But before doing so, in order to save the time of the Society, I may state generally the course of events which usually follow the operation. The catheter having been tied into the bladder, and secured by a plug to prevent the continuous discharge of urine, the patient is restored to his proper place in bed. On awaking from his chloroform sleep, he suffers so little pain or other inconvenience, as frequently to experience considerable difficulty in believing that the process has been accomplished. He then begins to

feel a pleasing consciousness of relief from what has long been a source of suffering and apprehension. He is tranquil and easily managed, not requiring opiates or any other ministrations, unless there should appear to be a greater degree of oozing from the wound than is convenient, in which case a piece of lint may be placed within the edges, and retained by slight pressure, until the bleeding ceases, which it usually does in a few minutes. The diet should be chiefly farinaceous, as animal food might cause thirst and restlessness; and mucilaginous drinks, according to the patient's taste, should be freely supplied, while wine and other stimulants are carefully withheld. At the end of forty-eight hours I have generally removed the catheter. But if the patient has not then begun to feel it uneasy, another day's delay, on the whole, seems advisable, since the escape of urine through the wound may thus be prevented, more frequently than in those cases where the instrument has remained only the shorter period.

A curious train of nervous symptoms occasionally present themselves, to the great consternation of all who have not previously witnessed them, or are unaware of their nature. They occur most frequently soon after the catheter is withdrawn, and appear to depend upon the urine resuming its natural course, but have also been observed at an earlier period. They have never, so far as I know, lasted more than thirty hours, and seldom continue above the half of this time. They consist of rigors, bilious vomiting, coldness of the extremities, suppression of urine, and delirium. They require no treatment, and do not seem to admit of being alleviated or curtailed by opiates, stimulants, or other means of remedy, requiring merely a little time for their disappearance, so that the only cordial of any service is a confident assurance on the part of the surgeon that there is no ground whatever for the slightest alarm or uneasiness. If the urine flows entirely through its proper channel, recovery may be considered complete in the course of a very few days, since the patient then makes his water in a full stream without pain or inconvenience, and with ordinary frequency.

having nothing to remind him of the operation except the superficial part of the wound, which soon contracts and cicatrises. When the urine escapes by the wound after the catheter is withdrawn, it generally continues to do so from one or two to eight or ten days, and then, diminishing to a few drops, speedily resumes its proper course. A full-sized bougie should be introduced once a week for three or four weeks, and then at more distant intervals, according to circumstances.

From the cases to which the attention of the Society is about to be directed, it will, I trust, appear: First, that stricture of the urethra occasionally presents itself in forms which resist the hitherto established means of treatment. Secondly, that strictures of the urethra, though deemed impermeable by surgeons of experience and reputation, may, through time and care, admit the introduction of instruments. Thirdly, that a free division of the thickened and contracted part of the canal, by external incision upon a grooved director, may afford complete and permanent relief in the most distressing and obstinate conditions of the disease. Each of the cases may not contribute evidence on all of these points, but the testimony of the whole will, I hope, be considered satisfactory.

Case 1.—About twelve years ago I was requested by the late Dr. Hay, of Edinburgh, to take charge of a gentleman suffering severely from stricture. It had existed upwards of twenty years, and, during the greater part of this period, admitted of such palliation from the use of bougies, as not to interfere materially with the patient's comfort, but latterly it had become aggravated to an extreme degree, so as to present symptoms of the most distressing character. Day and night the calls to make water were almost incessant; and in the intervals of his laborious and long-continued efforts to empty the bladder, the patient was constantly annoyed by an involuntary discharge of urine, with what discomfort may be more easily imagined than described. The stricture, which was anterior to the bulb, had contracted so as no

longer to permit the introduction of instruments. The tall and robust frame of the patient was emaciated to a shadow of what it had been, and he had become so sensitive that the slightest change of weather, especially in regard to humidity, greatly aggravated his complaint.

In these circumstances, I commenced my treatment by attempting to pass a bougie of the smallest size, and having soon succeeded in doing so, anticipated a satisfactory progress of the case. But, upon arriving at No. 5, I found the stricture obstinately resist any further dilatation, while the patient assured me that the degree already attained had afforded him no relief whatever. I then tried confinement to bed, and the retention of catheters in the bladder, with gradual increase of their size, until one of the largest capacity was introduced. During this process the patient felt so comfortable, and evacuated his water with so much more ease than he had long been accustomed to do, as fully to justify a belief that there would be no more trouble in future; but the very day after the instrument was withdrawn, the symptoms were renewed without any diminution. Soon after this, Mr. Liston happening to visit his friends in Edinburgh, I asked him to see this patient, and endeavour to suggest some more effectual means of treatment. He advised internal incision by the lanceted catheter, and I accordingly executed this procedure with such freedom that a full-sized bougie was passed immediately afterwards, without the slightest obstruction. As no benefit resulted beyond a partial relief of only one or two days' duration, I repeated the process still more freely, by using two catheters which cut on different sides, so as thus to make a double incision, but with no material difference in the effect it produced.

The patient, who had cheerfully submitted to this long course of abortive efforts to afford relief, now more anxiously than ever desired me to do something effectual, earnestly declaring, that next to recovery he would prefer an escape from his sufferings by death. I then told him, that in several cases of obstinate stricture in the anterior part of the canal I had overcome the difficulty by dividing the con-

tracted part through an external incision on a grooved director. He eagerly caught at this proposal, which I carried into effect in the manner that has already been described, and from that time to this, now more than ten years, the patient has enjoyed the most perfect health, local as well as general.[1]

Case II.—In 1844 Dr. Wickham, of Penrith, came to Edinburgh with a case of stricture, which had proved peculiarly obstinate and distressing. Although the patient was under 50, it had existed for twenty-seven years, and during the whole of this long period had never derived more than an imperfect degree of palliation from the use of bougies, notwithstanding their repeated and varied employment. Latterly, the symptoms having become much more severe, the assistance of a practitioner, deemed very skilful in the treatment of such cases had been required; but the patient returned home after two months' assiduous attempts to pass instruments, without ever having one introduced, and presented a new feature of alarming character in a tumour of the perineum, so prominent, circumscribed, and of such stony hardness, as to suggest serious apprehensions of carcinomatous degeneration.

Upon examination, I found that a full-sized instrument could be passed without pain or bleeding, down to the left side of the anus, fully an inch and a half beyond the probable or rather possible seat of stricture, which did not surprise me when I learned that the bougie had been used by pressing its point steadily upon the seat of resistance, so as to produce what has been called a "tunnelling" effect. Regarding the perineal tumour as the result of local irritation, I did not hesitate to make a free incision through its whole length. It was of almost cartilaginous firmness, but contained a few drops of matter in the centre, and it speedily disappeared after a free drain had been established. I then turned my attention to the urethra, and knowing that the mouth of the

[1] Case I has been described in the author's special treatise upon the subject of this paper.

tunnel must be anterior to the stricture, searched for the contracted orifice of the canal at a little distance before the bulb, where it was soon found, so as to permit the passage of a small catheter fairly into the bladder. At the end of six weeks the patient returned home, as I hoped effectually relieved by dilatation, but in the course of a few months he returned with the symptoms of stricture no less urgent than before.

I then introduced a grooved director through the contracted part, and freely divided it by external incision. In a few days the patient felt quite well, being, for the first time since the commencement of his disease, completely relieved from the uneasiness attending it, and greatly delighted with the facility no less than the efficiency of his treatment by the knife, instead of the tedious and abortive experience which he had had of the bougie. More than eight years have since elapsed, and, in reply to an inquiry which I lately addressed to Dr. Wickham, he says:—

"Agreeably to your request I have seen Mr. — to-day, and, so far as regards the stricture, I would say he is quite well. It is more than a year since any instrument has been passed into the bladder. On the last occasion I passed No. 13, with great ease."

Case III.—Mr. —, æt. 30, came from Ireland in December, 1849, to place himself under my care, on account of a stricture in the urethra. He had first become aware of its existence ten years before, from suffering retention of urine and requiring the catheter, which was introduced by a surgeon in Drogheda. From that time he had continued to labour under the symptoms of stricture, and sought assistance from a great variety of sources, but without ever having another instrument passed through the contracted part of the canal. In England and America attempts without end had been made unsuccessfully to accomplish this object; and upon one occasion, for nearly two months, Mr. Liston tried every other day in vain to introduce a bougie. At length the patient, despairing of relief, resolved

to endure the complaint, without any further attempt to remedy it, and continued to do so until the symptoms assumed that intolerable form in which the discomfort of incontinence is added to the difficulty of evacuation. He then committed himself to my charge.

In little more than a week I was able to pass a director through the stricture, which I divided by external incision upon the guide thus afforded. Recovery was delayed, by a little of the urine getting into the scrotal integuments, but was completed by the end of six weeks, and the patient then returned home, in the enjoyment of perfect health. On the 20th August, 1850, he wrote to me in the following terms:

"I promised to let you know how I was getting on, when I left Edinburgh, and as it is now a long time since I wrote to you, I have a good opportunity of knowing how matters stand. I am delighted to say, that there has never been the slightest obstruction or difficulty in making water, or in passing the bougie, which I do myself about once a month." In the following year, 1851, I wrote to inquire if the relief still continued, and received for reply:—"On my return last Saturday from England, I found your kind note, inquiring after my health. In answer, I am delighted to be able to tell you, that I have never felt the slightest return of my old malady. I sometimes pass the bougie, but it is only to satisfy myself, as I think now it is no use doing so. My health was never so good, and I can undergo more fatigue (which I do) than I ever could in my whole life."

This case affords encouragement to treat strictures deemed impermeable as accessible to the introduction of instruments, through their careful and persevering employment. It also illustrates the change in contractile disposition which results from dividing the parts concerned by external incision.

Case iv.—Lord —, æt. 35, married, and of correct habits in his mode of life, applied to me in the month of October last. He had suffered long from a stricture anterior to the bulb, and during the last five years had been under the treatment successively of three eminent surgeons in London,

who had employed dilatation by bougies, with the effect of affording partial but neither complete nor permanent relief, micturition being always more or less uneasy, and a corresponding degree of constitutional disturbance preventing the patient from ever having the enjoyment of perfect health. At length it was remarked, that whenever he made water a considerable swelling took place behind the stricture, and that after the expulsive efforts of the bladder ceased, a quantity of urine could be made to flow by pressure upon this part. As such a state of matters seemed to threaten the danger of extravasation it excited much alarm, and was deemed to require an aperture by incision. Instead of submitting to this proposal, the patient thought proper to request my assistance, and, regarding the dilatation of the urethra as an effect of the stricture, I resolved to strike at the root of the evil, by dividing the latter while I opened the former.

Chloroform having been administered, I introduced a small grooved director, and made an incision, as usual, in the middle line of the perineum, then pushed the knife through the dilated part of the canal into the groove, and carried it forwards so as to divide the stricture completely. A No. 8 silver catheter was next passed with perfect facility, and retained in the bladder for forty-eight hours. During the first thirty hours no uneasiness was experienced; but at the end of this time the train of nervous symptoms, to which I have adverted as no unfrequent occurrence, commenced, and continued for about the same period, so as to alarm the patient and his friends, although they did not occasion me the slightest anxiety. The urine did not escape at all through the wound, but, to the patient's great contentment, flowed copiously from the extremity of the urethra in a full stream, very different from that to which he had been so long accustomed. Convalescence was soon completed, without any interruption worthy of notice, and the recovery has been in every respect complete. I passed a full-sized bougie on two occasions within the first three weeks, and twice since then, with the interval of as many months, with the most perfect facility. Every trace of the disease has disappeared,

and the patient's general health is so much improved as to attract the notice of his friends.

From this case it appears that dilatation is not always sufficient for the remedy of stricture. The patient being a nobleman of the highest connections in London, and having the assistance of three surgeons, the most distinguished for skill and experience in the treatment of urethral disease, it must be supposed that the bougie was employed with every circumstance calculated to promote its efficiency. Yet we see that the disease, so far from being cured by these usual means, was very imperfectly palliated, and not prevented from producing effects of the most alarming kind. We also in this case see that a painless operation, followed by a few days' confinement, not only arrested the mischief in progress, but completely accomplished what a long trial of the ordinary treatment, in hands pre-eminently skilful, had failed to effect.

Case v.—Captain —, R. N., applied to me in January, 1851, on account of a stricture of which he gave the following history in writing:—"It was in the year 1829, when serving as a midshipman on board H. M. S. ——, in the Mediterranean, that I first observed symptoms of stricture, produced by gonorrhœa. Bougies were then introduced, and in a short time the complaint seemed to be overcome, and continued so up to 1833, when I was in the East Indies, on board H. M. S. ——, and suffered very much on boat-service, hunting pirates, &c., which exposed me to wet, and rapidly increased the complaint. I sought relief on board the ship but found none, as the medical men could not pass an instrument, and by their attempts generally induced complete stoppage of urine, when bleeding and the warm bath were the only means of any service. This state of matters continued for two years, until the ship was paid off, no instrument having been passed. Having got a little better from living quietly on shore, I sailed in May, 1836, as —— Lieutenant of H. M. S. —— for the Arctic regions, and up to the end of that year suffered little, except the incon-

venience of making water almost by drops. But the ice then suddenly broke up, exposing the ship to great danger, and obliging officers as well as men to expose themselves for considerable lengths of time to very low temperatures, even 80° below the freezing point, and upon some occasions to quit the ship. This had the effect of again sealing me up, and the old remedy (bleeding) was employed, as no one could pass an instrument. I suffered much until the end of the voyage in November, 1837, and then went into Chatham hospital, under the charge of Sir John Richardson, where I remained for six weeks with little or no relief, as nothing larger than No. 4 could be passed, and that produced very severe rigors at every attempt. Having learnt to use the bougie myself, I succeeded in keeping the passage open, until again, in the East Indies, in 1838, on board H. M. S. ——, it became so bad as to oblige me to return to England. In 1839 I sailed as —— Lieutenant of H. M. S. —— for the South Pole; during this voyage, which lasted until September, 1843, I suffered very much, and was obliged to quit the expedition a few months before to come home. I could neither make nor retain my water, which was constantly dropping away, so as to keep my clothes wet, while the temperature was from 20° to 30° below the freezing point. On my return to England, I went under Mr. ——, who succeeded in passing bougies up to No. 6, but no farther. He then used a catheter with a lancet-blade, but with little benefit, as in a month afterwards the passage was as much contracted as ever. I suffered severely from rigors during the time I was under Mr. ——'s care. Since then I have been three years upon the coast of Africa, and suffered so much from the disease, that upon two occasions lately I felt it necessary to decline the offer of service, knowing that had I accepted I should certainly have broken down."

Upon examination I found a slight contraction about three inches from the orifice, and a very tight stricture anterior to the bulb. Bougies were passed as usual up to No. 4, but then farther dilatation was resisted, and as the

patient had experienced no relief from the degree accomplished, I did not hesitate to divide the contracted part by external incision. For two days after the operation the patient felt perfectly well; but in the evening after the catheter was taken out, he became suddenly so ill, that I was sent for; I found him cold, nearly pulseless, and vomiting bilious fluid, in short, with every appearance of approaching dissolution. In the absence of other experience, these symptoms would doubtless have alarmed me; but knowing their true nature, I gave the confident assurance that all would be right in the course of a few hours. Accordingly, upon calling next forenoon, I found that the patient had breakfasted as usual, that his books were again in requisition, and that he was reading with a cheerful countenance. He made an excellent recovery, and at the end of a few weeks having received a letter from the Admiralty, offering him the command of an expedition to the Arctic Regions, at once accepted it. The stormy aspect of the political horizon delayed the necessary arrangements until too late for that season; but the patient's ready acceptance, after two refusals of a similar appointment, sufficiently shows the change in his feelings which had been produced by the operation. Wishing to know his present state, I wrote to him last month, and received for reply, that "he has not had a moment's uneasiness from the stricture."

Case vi.—Mr. —, æt. 27, from his earliest recollection had experienced undue irritability of the urinary organs, and deficiency of freedom in the urinary evacuation. In 1843 he went to the East Indies, and there suffered several attacks of gonorrhœa, which were followed by symptoms of stricture. In 1848, having returned home, he was exposed to severe cold while travelling in Ireland, and had retention of urine, which was relieved by warm baths and medicines without the use of a catheter. Next year a fresh attack of gonorrhœa was followed by an aggravation of the symptoms of stricture, which led to a long trial of instruments and subsidiary measures without success. Being in the service

of government, he then entered Haslar Hospital at Portsmouth, where between three and four months, to use his own expression, "innumerable attempts" were made with all sorts of rigid and flexible instruments, assisted by strict confinement to bed, repeated leeching and medical means, but all in vain, as, no more in the hospital than out of it, could the smallest bougie or catheter be passed through the stricture, which was in the usual situation anterior to the bulb. The patient then came to Edinburgh and placed himself under my care in August, 1850. At the very first attempt I passed a metallic bougie fairly into the bladder, and by the end of three weeks could introduce No. 10 without the slightest difficulty. He then went home, with instructions to have the instrument passed regularly.

Twelve months afterwards this patient returned, complaining that the symptoms of stricture were as troublesome as ever, and stating, that as his services had been transferred to London, he was desirous of being rendered fit for duty without the risk of further interruption. I again passed some small bougies on two different occasions; but finding that although there was neither pain nor bleeding at the time, severe rigors were afterwards experienced, and that the symptoms derived no alleviation, I advised the operation by external incision, and accordingly performed it as usual. Nothing occurred to impede recovery, so that by the end of three weeks the patient returned to London, in every respect perfectly well, and indeed making water with a degree of freedom which he had never known. Wishing to learn the present state of this gentleman, I requested my friend, Mr. Henry Thompson, to ascertain it for me, and received for reply, that No. 11 catheter passed with ease into the bladder.

This case shows that a stricture may be deemed impermeable without really being so, and will also, I hope, appear a convincing illustration of the advantages that may be derived from dividing by external incision a contraction of the urethra which resists the effect of dilatation.

CASE VII.—Captain —— began to suffer from stricture in 1822, when in the cavalry depôt at Maidstone, soon after entering the army. The military surgeon could not pass any instrument; he repaired to London and placed himself under the care of the late Mr. Earle, who, in the course of five or six months, after trying various plans, at length succeeded in dilating the canal to its full extent. The patient then went to the East Indies, and during the three years which he passed there, as well as after his return home, regularly introduced bougies. This, however, did not protect him from occasional attacks of retention, which required the catheter, and were observed to happen most frequently the day after he had passed the bougie. In 1838, finding that the disease was gaining ground, he applied to Mr. ——, and remained under his care for a month, but without deriving any permanent advantage, so that it was necessary again to request his assistance in the same year, when the stricture was cut by internal incision. He then went to the West Indies, and continued to pass bougies regularly. In 1844 he married; in 1848 the disease assumed its formidable condition of extreme irritability. The calls to make water were almost incessant, and produced efforts so ineffectual, that a small catheter was required to empty the bladder three or four times a day. Severe pains of the perineum and limbs prevented every sort of active exertion, while the state of suffering and oppression frequently required confinement to bed for weeks. The patient, at the end of two years passed in this miserable state, feeling thoroughly disabled for the enjoyments and duties of life, resolved to try the effect of my treatment. He accordingly came to Edinburgh in July, 1850, and as the case seemed very suitable for division of the stricture, I performed this operation without delay. The patient made a good recovery, and from that time forward has never had retention of urine, frequent calls to make water, pain or difficulty in doing so, or, in short, any of his former ailments. He enjoys the best of health, and is able to take the most active exertion. The bougie is still passed once a week by the patient, but more

as a precautionary measure than from any feeling of its being required.

This case shows that dilatation affords no security against the progress of a stricture, from its milder form to that of the most distressing character. It also affords an example of complete and permanent relief from dividing the stricture by external incision, under circumstances which rendered any of the other means of treatment hitherto proposed utterly hopeless, and indeed inadmissable. Nearly three years have now elapsed since the operation was performed, and I had lately the pleasure of receiving from the patient the satisfactory account of his condition which has just been stated.

Case VIII.—In the month of July last, Mr. James Miller, practitioner in Edinburgh, asked me to see a very distressing and perplexing case. The patient, Mr. —, æt. 41, had begun to suffer from a stricture of the urethra thirteen years ago, but paid little attention to it until four years since, when about to be married. Mr. Miller then requested the assistance of Dr. Duncan, lately Senior Ordinary Surgeon of the Royal Infirmary, who soon succeeded in passing instruments, and at the end of little more than two months had fully dilated the canal. Twelve months after marriage the symptoms of stricture were again so troublesome, that the aid of Dr. Duncan was requested with the effect of again dilating the urethra, so as to admit bougies of the largest size. But upon this occasion, instead of relief, there was an aggravation of the complaint. In November, 1851, the state of matters became so bad, that from this time to May, 1852, the catheter was required twice a day for drawing off the water, the patient being confined not only to the house, but chiefly to bed; he then went to the country for some weeks, but returned without any improvement, and twelve months nearly having then elapsed since he had been laid aside from the active duties of life, and rendered unable to take any charge of his business, though having every inducement and disposition to do so, he became very desirous to obtain some effectual relief.

In these circumstances I found him, lying in bed with an emaciated aspect, and care-worn, anxious countenance, expressive of suffering and apprehension, complaining of constant pain and uneasiness about the perineum, aggravated at every attempt to make water, which passed by small quantities, in drops or in a slender stream. On examining the urethra, I found that a moderate-sized instrument, and even one of the full size, could be introduced, but not without encountering a little difficulty, and requiring some guidance at the seat of stricture, which was anterior to the bulb. According to the opinions hitherto entertained generally, so inconsiderable a degree of contraction would not have been deemed sufficient to account for symptoms so obstinate and severe. But having known complete and permanent relief afforded under similar circumstances, by external incision through the seat of stricture, I advised this mode of procedure, and carried it into effect with the most satisfactory result. The patient was at once entirely freed from all his uncomfortable feelings, quickly regained his health and strength, and ever since has been perfectly well. I have on three occasions, at distant intervals, passed a full-sized bougie, without the slightest catch or obstruction, and the patient finds it difficult to express his sense of obligation to the treatment by external incision, which so speedily transferred him from a bed of helpless and hopeless misery to the full enjoyment and usefulness of vigorous health.

This case illustrates the progressive disposition of stricture, and the inefficiency of dilatation either to prevent or to remedy the distressing symptoms which are occasionally presented by the disease. It also affords an example of the immediate, complete, and permanent relief which may be obtained under such circumstances from the external incision.

Case ix.—Mr. —, æt. 23, applied to me in October last. He stated that from the earliest period within his recollection he had experienced difficulty in making water, and particularly noticed this infirmity upon first going to school, from

taking so much longer time than the other boys. At ten years of age he suffered from retention of urine, requiring the warm bath and confinement to bed. Next year he had a more severe attack, requiring the catheter, which one medical man failed and another succeeded in introducing. In every year subsequently, and generally about the autumnal season, he suffered from complete retention, while in the intervals he made water with great difficulty, in a very small stream. Indeed, from the age of fifteen, he said, that it had always been necessary for him to empty the bladder in the same position as when evacuating the bowels, and then only through laborious efforts, aided by pressure on the perineum at the seat of stricture, which was just below the scrotum. At length the suffering became so great and incessant, notwithstanding the use of small bougies, which indeed aggravated the symptoms, that he resolved to come from Canada to place himself under my care, and accordingly crossed the Atlantic for this purpose.

Feeling sure, from the history of this case, that effectual relief could be afforded only by complete division of the stricture, I administered chloroform, passed a small grooved director through the contracted part of the canal, and divided it freely by external incision. The patient was at once relieved from the distress he had so long experienced, and in the course of a few weeks might have returned to America, had he not feared a relapse. I saw him within the last fortnight, and although four months had elapsed since the introduction of any instrument, I passed No. 10 without the slightest difficulty, so that with ordinary care the recovery may now, I think, be deemed permanent.

From this case, and others which have fallen under my observation, I am inclined to suspect that the occurrence of stricture before the age of puberty, and independently of sexual disease, is not so very rare as might be concluded from the silence of surgical writers on the subject.

Case x.—Lieutenant —, of H. M. — Regiment of Foot, in 1841 contracted a gonorrhœa, which was treated after a

somewhat peculiar fashion, by the introduction of large bougies, and the cold shower-bath, with the effect of stricture being induced, and manifesting its presence by the usual symptoms, within the following eighteen months. He then went to India, and, during six years which he passed there, was under the care of five different medical men, who tried catgut bougies long and assiduously, without being able to pass them, and also caustic with little better success. In the course of these ineffectual attempts to afford relief, his general health had become impaired, and in 1849 he was sent home, by the decision of a Medical Board. He arrived in England in March, 1850, and placed himself under the care of an eminent surgeon, who during the next eight months passed bougies twice a week, but, to use the patient's own words, "the stricture, like India-rubber, always went back to about the same size" (No. 3 or 4), and the scalding, from which he had never been free since 1841, continued. Next year his army surgeon passed instruments occasionally, without affording any benefit, and towards the conclusion of it, finding the state of matters worse than ever, inasmuch as incontinence began to be experienced, in addition to the difficulty of evacuation, he applied to Mr. ——, of London, and remained under his care from December to the following April, 1852. In the first instance no instrument could be passed, but caustic having been employed every two or three days for a month, under chloroform, No. 3 catheter was introduced, and retained in the bladder, and then Nos. 5, 7, 9, 11, and 12, with an interval of two days between each. Violent irritation succeeded the introduction of the last instrument, and prevented any other from being used for a fortnight, when No. 6 was passed with difficulty, and after a month or six weeks' farther trial with bougies, No. 9 was the utmost extent that could be reached, while the symptoms remained unrelieved. The patient then rejoined his regiment, and had bougies introduced regularly without any benefit, up to the end of October, when the attacks of ague, from which he had long suffered occasionally, became nearly constant, so as to confine him entirely to

bed. In these circumstances, the surgeon wrote to ask me if I would undertake the treatment of the case, and as I readily agreed to do so, the patient came over from Ireland, and arrived in Edinburgh on the 22d of December.

I found him lying in bed, much exhausted by his sufferings on the passage, and still labouring under one of the aguish attacks from which he was seldom free; the urine was very turbid, and excessively loaded with mucus, which formed a large glairy mass at the bottom of the vessel containing it, and all the symptoms of stricture were present in an extreme degree. On the 24th, I divided the contraction by external incision, with some difficulty, from the excessive thickening and induration of the textures concerned, which resembled cartilage much more than the urethra in its ordinary state. After the operation there was no return of ague or the slightest tendency to rigor, and in the course of a few hours the urine became clear, and free from mucus. The catheter was removed at the end of two days, when a portion of the water escaped by the wound, but gradually diminished in quantity, and ceased entirely within a fortnight, when the patient might be considered, in every respect, as perfectly well. He had not been confined beyond three days to bed, and instead of finding the time pass slowly, at a distance from his friends and occupation, declared that he never felt weary in enjoying the delightful sensation of relief from the scalding and other sorts of uneasiness which had been experienced, without intermission, for the last twelve years. On the 12th of January, as his regiment had been ordered to embark for foreign service, he departed for Cork, and there finding that sufficient time remained for the purpose, proceeded to London, and thence returned to Cork, in the midst of frost and snow, without ever having a bougie passed; then he engaged in all the fatigue and turmoil of departure, and sailed in a different transport from that which conveyed the regimental surgeon. This was, indeed, a severe ordeal for the operation, and if, as I hope and trust, it has stood the trial, the result will be all the more satisfactory. But the case is important in another point of view, by showing

the inefficacy of dilatation and caustic in the hands of gentlemen who, from their position, must be supposed to have employed these means in the most perfect manner. It also shows the progressive nature of the symptoms, and the complete relief immediately afforded by division, in circumstances which, I believe, might have been justly deemed hopeless under any other mode of treatment.

Case xi.—Captain —, æt. 45, retired from the army, had suffered long from stricture, and had been repeatedly under the care of a person practising in London. Time after time he was dismissed with an assurance of complete and permanent relief. Until becoming tired out, he consulted Mr. ——, of the — Hospital, who having examined the urethra, gave it as his opinion that the use of instruments could be of no service, and after a month's trial of medical treatment, dismissed the patient, with advice to endure what could not be cured. From January to December of last year he endeavoured to comply with this suggestion, but feeling very uncomfortable, and still clinging to the hope of relief, he came from one of the southern counties of England and placed himself under my care.

I found that the stricture, which was in the usual situation anterior to the bulb, possessed the irritable disposition that occasions spasmodic contraction, and prevents any advantage being gained by dilatation. The patient could never make water with freedom, or be sure of making it at all, from attacks of retention, which admitted of relief only through the introduction of an elastic catheter of the smallest size. In these circumstances I did not hesitate to divide the stricture by free external incision, and in less than a fortnight had the satisfaction of seeing that no vestige of uneasiness remained. In a letter just received (14th April), he says, "that although upon two occasions some difficulty was experienced in passing the bougie, he has passed through the winter with the greatest comfort, moving about in perfect security, and living much in the open air in spite

of keen frosts and razor-edged gales from the east. No disturbance, no retention, no distressing 'ennui de pisser.'"

Case xii.—In 1849, Mr. —, æt. 60, applied to me on account of a stricture anterior to the bulb, from which he had suffered more than thirty years. During this long period Mr. Liston, and other practitioners, by the use of bougies, had palliated the symptoms, but never completely relieved them, and latterly they had become much more troublesome than ever. The urine was at all times passed with extreme difficulty in a small dribbling stream, or by drops, and the patient was so liable to retention, that he never felt secure without having a slender flexible catheter in his pocket. I tried dilatation, but could not carry it beyond No. 4, and finding that the patient derived no benefit from this degree of improvement, proposed to divide the stricture by external incision, to which he readily assented. On the following day, after the catheter was removed, the urine resumed its natural channel, and flowed so freely, that the patient felt at once completely free from all the annoyance and apprehension he had so long experienced. I desired him to call upon me occasionally to have a bougie passed, but this he neglected, and when he at length returned in the course of the following year, I found that the contraction was re-established, so as to present the same symptoms and resistance to dilatation as before the operation. I therefore proposed to repeat it, and in doing so took care to divide the urethra much more freely than on the former occasion. Since then, although more than two years have elapsed, there has been no return of the disease, and every third or fourth month I have passed a bougie of the full size without any difficulty.

This case shows the efficacy of division in subduing the resistance which is proof against dilatation, and also illustrates the advantage of making a free incision through the contracted part of the urethra, since had the second operation not been performed, it might, and no doubt would, have

been said, that in this instance the procedure had proved insufficient to afford permanent relief.

Unwilling to fatigue the attention of the Society, or occupy their time unnecessarily, I will add no more cases, trusting that the positions which I undertook to establish may appear sufficiently supported by those that have now been related. They are selected from private practice, as admitting of more easy and satisfactory reference than the subjects of hospital experience; and if circumstances had seemed to require it, could have been greatly increased. It will be observed that I have not now, any more than heretofore, presumed to throw blame on my brethren for the use made by them of the means hitherto devised for the remedy of stricture; the extent of my allegation being that these means are not always sufficient for the purpose, and that on some occasions dividing the contracted part by external incision may be added to them with advantage. It is quite possible that I may have attached undue importance to the operation in question; but I venture to hope, that whatever degree of confidence it may ultimately be destined to acquire, this Society will, at least, give me the credit of good intention in bringing it under their notice.

FURTHER RESEARCHES

ON THE

PATHOLOGY OF PHLEGMASIA DOLENS.

BY

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More than a century and a half had elapsed from the time when the disease termed, l'enflure des jambes et des cuisses de la femme accouchée, depôts laiteux, and phlegmasia dolens, was first described by Mauriceau, and an attempt was made to discover, by morbid anatomy, the proximate cause or the true nature of the affection. Before the publication of the memoirs of M. Bouillaud, the late Dr. Davis, and M. Velpeau, various hypotheses had been advanced respecting the cause of the swelling in the lower extremities of puerperal women, but they were mere speculations, unsupported by facts, and inadequate to account for the symptoms. The cases and dissections related by these authors first threw light on the real nature of the disease, and showed that it consisted in an inflammation of the trunks and principal branches of the veins of the lower extremities.

My papers, published in vol. XV of the 'Medico-Chirur-

gical Transactions,' contained a report of thirteen cases of phlegmasia dolens, in six of which the actual condition of the iliac and femoral veins was ascertained by dissection. From this I was led to infer, that inflammation of the iliac and femoral veins gives rise to all the phenomena of that disease in puerperal women, and "that in phlegmasia dolens the inflammation commences in the uterine branches of the hypogastric veins, and subsequently extends from them into the iliac and femoral trunks of the affected side."

The second of these papers contained the histories and dissections of three cases of cancerous ulceration of the os and cervix uteri, in which there was inflammation of the internal, common and external iliac and femoral veins, and all the characteristic symptoms of puerperal phlegmasia dolens.

Mr. Lawrence, in vol. XVI of the Transactions of this Society, has recorded a similar case, and Mr. Holberton two cases, in which crural phlebitis followed ulceration of the mucous membrane of the intestines.

According to the concurrent experiments performed on dogs by Pirigott, in 1839,[1] and Reumert, in 1840,[2] the tendency to the extension of inflammation in veins otherwise healthy is not so great as is commonly supposed. The action of chemical and mechanical irritants remained for the most part limited to the vein, on which the experiment was made, and extended further only when the vein was at the same time laid bare, or when the foreign body was kept in continued contact with it. Whether inflammation of venous trunks admits of being excited from constitutional causes independently of local irritation, Stanius considers doubtful, after having collated and tested all the facts bearing on the subject.[3]

[1] Ann. der Chirurg. Abth. des Clinikums von Dorpat Zweiter Jahrg.; Dorp. 1839.

[2] De Symptomatibus Inflammationis Venæ Cavæ Diss. Inaug.; Havn. 1840, p. 46.

[3] Ueber Krankhafte Verschleissung grössen Venenstämme; Berlin, 1839.

A series of experiments on the lower animals similar to those now described, have recently been made in this country, and a paper on Phlegmasia Dolens has been read to this Society during the present session, not founded on actual observation of the disease, as it occurs in the human subject, but upon these experiments on the veins of the lower animals, in which phlegmasia dolens has never been observed.

The object of the present communication is to submit to the consideration of the Society the observations which I have made during the last twenty-four years on inflammation of the crural veins.

Case xiv.—A lady, æt. 26, was delivered prematurely of her first child, on the 19th of June, 1831. The placenta being retained beyond the usual period was extracted artificially. In a few days, pain in the region of the uterus, with fever, and great prostration of strength, took place. After the application of leeches, the tenderness of the hypogastrium disappeared, but the pulse continued frequent till the end of the third week, when a painful sense of tension was experienced along the brim of the pelvis, on the left side, and this was speedily followed by phlegmasia dolens in the left lower extremity.

On the 21st July, about a month after delivery, I saw the patient with Mr. Cleland, of Rock Hill, Ratcliff. The pulse was 150, and feeble. There was constant nausea, vomiting, and diarrhœa. The tongue was of a dark brown hue. The countenance and whole surface of the body was of a yellowish dusky colour. The respiration was hurried, with frequent cough and expectoration; there was great debility and occasional delirium. The whole of the left lower extremity was swollen to nearly double the size of the other. The femoral vein, exquisitely tender on pressure, felt large and hard in the upper part of the thigh, and there was fulness and tension above Poupart's ligament, in the situation of the iliac veins. The foot and ankle pitted upon pressure; but the integuments of the thigh

were hot and tense, and did not retain the impression of the finger.

On the 22d, there was great prostration of strength. Pulse 160. Respiration laborious. Tongue dry and brown; diarrhœa, and vomiting. She was conscious at intervals, and then complained of great pain along the inner part of the left thigh, and in the ham. The abdomen was tympanitic.

Death took place on the 24th; and I examined the body with Dr. Sims and Mr. Cleland, on the 25th.

The uterus had sunk down into the pelvis, and was as much reduced in size, as it usually is four weeks after delivery. The peritonæum at first sight appeared every where healthy, but on closer inspection, an adhesion by means of false membrane was found to exist between the posterior part of the uterus and rectum. More than a pint of fluid was contained between the uterus and rectum. The peritonæal and muscular coats of the fundus and body of the uterus were so soft as to be readily torn with the fingers, and of an inky black colour. On laying open the uterus the placenta was found to have adhered to the posterior and inferior part of the uterus; and the branches and trunk of the left internal iliac vein were all filled with purulent fluid, and their inner surface lined with a false membrane of a blackish colour. The coats of the common, external iliac, and femoral veins to the middle of the thigh were all thickened, and their cavities filled with soft coagula of lymph and pus. The vena cava to about two inches below the hepatic veins, was completely blocked up with a coagulum of lymph, which partially adhered to the inner surface of the vessel. Several glands in the vicinity of the vena cava and iliac veins were in a state of suppuration. The coats of the left internal iliac vein at its termination in the common iliac were in a soft shreddy state. The right common, internal and external iliac and femoral veins, were all in a healthy condition.

Case xv.—On the 27th of January, 1832, a patient of

the British Lying-in Hospital was delivered after a natural labour. Obscure febrile symptoms soon took place, without any uterine pain, and I was not called to see her till ten days after her confinement, when the pulse was 130; the tongue of a dark glossy red colour; the lips parched; the countenance of a dusky yellow colour; tremors of the muscles of the face and extremities, with occasional delirium. There was no pain, tension, or swelling of the hypogastrium; but there was exquisite pain on pressure along the course of the iliac vessels, and down the inner part of the thigh, on the left side, and the whole of the left lower extremity was much swollen, hot, tense, and shining.

On the 13th, the pain in the course of the iliac and femoral vessels had been relieved by leeches and fomentations. The pulse was 140. The tongue dry and brown. The conjunctivæ of both eyes had suddenly become intensely red and swollen, and the vision impaired, if not lost. The right knee-joint had become exquisitely painful when moved; but it was neither red nor swollen. A dark coloured gangrenous spot had appeared over the sacrum, and she died on the 18th.

The uterus had subsided into the pelvis, and there was no trace of disease perceptible in the peritonæum. Both spermatic veins were healthy. The coats of the left common, external iliac, and femoral veins, deep and superficial, were all thickened, and their cavities plugged up with firm coagula. The same was the case with the epigastric vein, and circumflexa ilii. The glands in the vicinity of these veins were enlarged, red, and vascular, and closely adherent to the cellular membrane and outer surface of the vessels. The vena cava, to a short distance above the entrance of the left common iliac vein, had its coats thickened, and a soft coagulum of lymph adhering to its inner surface. The uterine, vaginal, gluteal, and most of the other veins, which form the left internal iliac, were gorged with pus, and lined with false membranes of a dark colour approaching to black. The uterine branches of the right internal vein were also filled with pus and lymph; but the inflammation had not

extended beyond the entrance of the trunk of the vessel into the common iliac, and the right common external iliac and femoral veins were all in a healthy condition. In the muscular tissue of the cervix uteri on the left side, was a cavity which contained ʒiij of purulent fluid. The veins proceeding from this part of the cervix were filled with pus, and the muscular coat of the body of the uterus was as soft as lard. The conjunctivæ, which before death had been red and swollen, were now almost colourless, scarcely a vessel containing red blood being discernible. The deeper-seated parts of the eye were not allowed to be examined.

Case xvi.—Several years ago, I was requested to see a young woman in labour, who resided near the Seven Dials, in whom it became necessary, from the presence of a large fibrous tumour in the walls of the uterus, to have recourse to the perforator and crotchet. In a few days, rigors, rapid pulse, and other symptoms of uterine phlebitis, occurred, and subsequently, phlegmasia dolens in the left lower extremity. Death took place, and on examining the body, the fibrous tumour was found in a softened broken-up condition. The left internal iliac vein filled with pus and lined with false membrane, and the coats thickened, and coagula of blood distending the common and external iliac veins.

Case xvii.—A young healthy woman, a patient of the Southwark Lying-in Institution, was delivered, after a natural labour, on the 21st October, 1832. Rigors, uterine pain, headache, and fever, soon took place.

On the 31st she complained of great tenderness in the situation of the symphysis pubis, and in the right groin, extending along the course of the femoral vessels, which felt hard and cord-like, beneath Poupart's ligament, for a space of three inches down the thigh. The limb greatly swollen and œdematous, was free from discoloration except below the ham, where it was of a dark hue, from distension of the saphena veins. The pulse became very rapid, diarrhœa and gangrenous inflammation of the parts covering the

sacrum, and the right outer ankle and foot, and death took place soon after. The bones of the pubis were found separated, a quantity of pus in the pelvis, and the right femoral vein plugged up with fibrine, and the saphena major filled with pus. Permission could not be obtained to examine the state of the internal iliac veins.

Case xviii.—On the 31st December, a young woman died in the St. Martin's Parochial Infirmary, five or six weeks after her confinement. Before pregnancy, she had suffered from cough dyspnœa, and other symptoms of tubercular phthisis. The symptoms became aggravated after delivery, and it was supposed before a post-mortem examination was made, that she had died from chronic disease of the lungs. Before her death she had also suffered from obscure symptoms of uterine inflammation, for which leeches had been applied to the hypogastrium. I examined the body with the late Mr. Gosna, and received from him the report of the symptoms.

The inferior lobe of the lungs on the right side was hepatised and encrusted with lymph. On the left side there were extensive adhesions, not recent. There was no peritonæal inflammation. The uterus had descended into the pelvis, and was of the usual size, four weeks after delivery. The left fallopian tube adhered to the uterus low down behind. The fundus uteri and posterior part of the body were of a peculiar yellow colour. Two patches of a blackish colour were observed near the cervix, where the corpus fimbriatum adhered. There was a small abscess in the left broad ligament. The sinuses in the cervix and body of the uterus contained pus. The branches of the internal iliac veins were all filled with a soft, yellowish coagulum of lymph. This was traced to the common and external iliac, which were both filled with coagula of soft lymph, in the centre of which was a thin matter like pus. About one inch above the entrance of the internal into the common iliac the coagulum ended abruptly, and a dark coagulum of blood, which did not adhere closely to the vessel, occupied

the greater part of the remainder of the common iliac. A coagulum filled a great part of the vena cava. Cough and scanty viscid sputa, and pain in the chest. She was ordered calomel, and opium, and leeches, and a blister to the chest. Rigors and perspiration came on, also diarrhœa and vomiting, and she sank and died.

Case xix.—On the 12th October, 1842, I examined the body of a woman who had suffered from phlegmasia dolens in both lower extremities, the left having been first and most severely affected. She had been under the care of Mr. Brookes, of Bedford Street, and Mr. Jones, Soho Square, and before death, she had suffered chiefly from great debility, fever, constant sickness, and diarrhœa. All the pelvic viscera, blood-vessels, and nerves were removed. There was no trace of peritonæal inflammation about the uterus, and the intestines were perfectly sound. All the veins of the cervix uteri on the left side were filled with pus and lymph, and coated with false membranes. The left internal common and external iliac and femoral veins were in the same condition. The coats of the vena cava were greatly thickened as high as the diaphragm, and a rough false membrane adhered to the whole of its inner surface. Within this was a soft yellowish substance, which washed away like a mixture of pus and lymph, with coagula of blood. A quantity of purulent matter escaped from the vena cava when it was opened near its bifurcation; and at this point there was a firm union between the vein and the last lumbar vertebra and cartilages. The right common external and internal iliac and femoral and saphena veins, were all destroyed by inflammation, though less disorganised than on the left side. The left spermatic vein from the uterus to its termination in the renal vein, was thickened and lined with false membranes, and filled with coagula of blood. The cellular tissue of the limb was filled with serum.

Case xx.—A young lady was delivered of her first child, after a protracted labour, at the end of June, 1844. In the

course of a few days uterine inflammation took place, which was relieved by antiphlogistic treatment. Six weeks after a second attack of uterine inflammation took place, which was speedily followed by phlegmasia dolens in the left lower extremity; from this she was likewise relieved. In August she had a violent attack of peritonitis, by which she was nearly destroyed. At Ramsgate, where she had gone for the sea air, another attack took place, which reduced her to a state of great debility.

On the 27th October, after a slight irregularity of diet, was seized with violent pain and vomiting, collapse soon took place, and she died on the 30th. The body was examined on the 1st November.

Extensive old adhesions were found between the peritonæum and uterus, and quantities of recent lymph. All the veins of the uterus on the left side were filled with coagula. The coats of the internal, common and external iliac veins, and lower part of the vena cava, were all thickened and plugged up. There was nothing unusual observed in the limb.

Case xxi.—Mrs. G— was delivered with the forceps on the 10th October, 1844, after having been upwards of forty-eight hours in labour. In a few days fever and dyspnœa took place.

On the 6th November the symptoms were more severe, and the vision of the right eye was suddenly lost.

On the 8th the iris was covered with lymph, and the eye was greatly swollen. There was severe pain in the right shoulder-joint, and in the right hand and fingers, which were stiff and swollen. The whole of the left lower extremity was affected with the swelling, and other symptoms observed in phlegmasia dolens, and there were two large purple spots on the inner part of the left leg; pulse 130.

On the 10th the symptoms were aggravated, and a large gangrenous spot had formed over the sacrum.

She died on the 16th, and I examined the body on the 17th.

There was no inflammation of the peritonæum. The left internal iliac vein, through its whole course, was lined with a thin false membrane, which closely adhered to the inner surface of the vessel, and near the uterus its cavity was filled with pus. All the veins on the left side of the os and cervix uteri, and those of the vagina, were inflamed and full of purulent fluid. The coats of the left common iliac were thickened, and its cavity filled with a soft pultaceous mass, the outer surface of which adhered to the vein. A coagulum of blood, three inches in length, was contained in the lower part of the vena cava which had not been inflamed. The left common iliac vein, the superficial and deep femoral, and the saphena veins, were all filled with firm coagula of blood; but the coats of none of these vessels presented the alterations of structure usually observed after phlebitis. The cellular membrane of the whole of the left lower extremity was filled with serum.[1]

Case xxii.—This case occurred in St. George's Hospital, and I am indebted to Dr. Wilson for the following details, and permission to place the preparation on the table of the Society. A specimen of fibrinous coagula in the veins, consisting of a portion of the inferior vena cava, the right common iliac veins, and the left, and also with its bifurcation into the internal and external iliac veins. They all, except the right common iliac, contain quantities of coagula of various colours and consistency. The entire vena cava, inferior as high, but not higher than the beginning of the connections with the liver, a portion of which is visible in the preparation, is affected. At the upper part the coagulum was less abundant, firmer and paler, and the lining smooth surface on which it lay was whiter. But in the lower parts, the venous tunics are equally thickened, and the coagula are in greater abundance, giving a stain to the walls of the vein. The various veins were firmly adherent by consolidated areolar tissue, surrounding them to their respective arteries and contiguous structures, and the lym-

[1] See paper on Ophthalmia in Puerperal Women.

phatic glands of the iliac, femoral, and lumbar regions were greatly enlarged. The right common iliac vein is laid open, being quite healthy, and without contents. In addition to the products visible in the veins, there also existed a quantity of dirty-looking greyish-red puriform substance, which, owing to its fluidity, has been washed away. Along with this state of things was found also great distension of the lining surface of the spermatic veins, as well within as without the broad ligament; the left hypogastric and uterine veins were in a similar condition, but did not contain any coagula. On examining the veins within the broad ligaments of the uterus, on the left side, there was found a cavity, containing nasty brown puriform fluid, but apparently not connected with any venous trunk. The veins on the right side were healthy. On examining the uterus, the muscular structure was found healthy, but the mucous lining was destroyed, displaying a free roughened surface of an ash-grey colour, and in the right corner of the uterus was a portion of surface to which a placenta had evidently been attached. The mucous membrane of the os uteri and vagina was flaccid and congested, as well also as the uterus. There was also found degeneration of the kidneys and recent pleurisy, with bronchitis and secondary lobular pneumonia in various stages of progress. Some parts having softened and advanced to the formation of purulent matter; others yet presented the light colour and firm consistency, surrounded by dark congested parenchyma peculiar to the early stage. Throughout the body the blood was very fluid, and the structures generally softened.

The subject of this attack was Elizabeth Gould, æt. 22, a patient of Dr. Wilson's, who died July 11th, 1851, one month after a premature confinement, being at the time only six months pregnant. Parturition was succeeded by abdominal pain and constipation, subsequent diarrhœa and rigors, with pain and swelling in the calf of the leg, came on, and this extended up the thigh. The abdominal pain subsided. On admission into the hospital, the tongue was coated, the pulse quiet and feeble, but the abdomen was

soft and free from pain. The thigh was very tender on pressure, and swelled. There was dyspnœa, cough, and scanty viscid sputa, and pain of the chest. She was ordered calomel, and opium, and leeches, and a blister to the chest. Rigors and perspirations came on, also diarrhœa and vomiting, and she sank and died. In this instance it is remarkable that the state of the veins should have begun, as was doubtless the case, in the uterine veins, and yet they themselves should be so slightly affected; and also, that for some cause or other, the iliac and femoral veins on one side are entirely unaffected, the affection having crept upwards high into the inferior vena cava, and low down into the veins of the other side.[1]

From the whole of the facts now adduced, it may be again "inferred that inflammation of the iliac and femoral veins gives rise to all the phenomena of phlegmasia dolens, and that the inflammation commences in the uterine branches of the hypogastric veins, and subsequently extends from them into the iliac and femoral trunks of the affected side."

The following cases furnish additional evidence in favour of this conclusion, though, in consequence of the recovery of the greater number of the patients, an opportunity was not afforded of determining, by dissection, the actual condition of the crural veins.

Case xxiii.—Mrs. W—, æt. 26, was delivered, after a protracted labour, of her first child, on the 9th of July, 1830. In less than twenty-four hours great tenderness of the abdomen came on, with nausea, retching, headache, restlessness, and rapid pulse. Dr. Merriman saw her with me, and venesection, leeches, calomel, and opium were freely employed.

On the 14th the pain in the region of the uterus had disappeared, but the pulse was 140.

On the 16th the pulse was 126, and the gums were affected with the mercury. She complained of pain in the

[1] Post-mortem Book, p. 185.

left iliac region, and along the femoral vein, increased by pressure, and there was an unusual fulness in this situation.

August 28th, 1830.—During the last month the pulse has never been below 100, and frequently 110. There have been slight febrile attacks in the evening, sense of coldness followed by profuse perspirations. The pain in the left side of the hypogastrium has never wholly disappeared, and it has been accompanied with uneasiness over the whole abdomen, and distressing flatulence. On examining the left iliac region it is found to be fuller and harder than natural, and sore on pressure. Along the brim of the pelvis, in the situation of the external iliac vein, there is felt a considerable hardness and swelling; this can be distinctly traced to the distance of an inch or two under Poupart's ligament, where it is very painful on pressure. There is also tenderness along the inner surface of the thigh, but the femoral vein cannot be traced down the limb, and there is no swelling of the superficial veins. The limb afterwards became swollen but in a slight degree, and it appeared probable from the symptoms, that the inflammation had not extended beyond the iliac veins. For a very considerable period the thigh remained contracted upon the trunk and could not be extended.

Case xxiv.—On the 17th January, 1831, I was called by Mr. Anson to see a puerperal patient residing at 12, Dorset Street, Manchester Square, who was suffering from phlegmasia dolens in the left lower extremity. The whole limb was swollen, tense, hot, and colourless. The femoral vein could not be distinctly felt, but there was exquisite tenderness on pressure in the left iliac fossa, and along the course of the femoral vein to the ham. There was no pitting on pressure in any part of the limb, and the power of moving it was entirely lost. Pulse 120. Tongue foul. Anorexia. The delivery had taken place three months before. It was observed that the pulse continued unusually quick from that time till the appearance of the swelling of

the limb, which took place on the 16th. The intumescence of the lower extremity was preceded by tenderness of the hypogastrium and left groin, sickness, and remarkable depression of strength and spirits. The symptoms were soon relieved by the repeated application of leeches along the course of the iliac and femoral veins, fomentations, and other means.

Case xxv.—Mary Eggius, æt. 19, No. 20, Little Mary-le-bone Street, a patient of the Middlesex Hospital, under the care of Dr. Hugh Ley, was delivered of her first child on the 26th of November, 1831. The labour was natural, and there was no enlargement of the veins of the lower extremities during pregnancy. On the sixth day after delivery she was attacked with rigors, vomiting, great sensibility of the hypogastrium, and suppression of the lochia. On the ninth day pain was experienced in the left groin, and soon after a swelling took place in the calf of the leg and ham, which gradually extended over the whole limb. For seven weeks the extremity continued swollen to double the size of the other, hot and painful, and incapable of being extended or moved.

January 25th, 1832.—The limb is still larger than the other, and pits. She suffers from occasional severe attacks of pain in the lower part of the abdomen, and along the course of the femoral vessels. The superficial veins of the lower part of the abdomen and upper part of the thigh are enormously enlarged. Around the ankles there are large clusters of varicose veins. Blisters, which produced extensive ulcerations, were applied to the calf of the leg without benefit. The general health is much impaired, and she has only partially recovered the use of the limb.

Case xxvi.—On the 3d December, 1834, I saw a lady, with Mr. Powell, at 66, Grove Street, Camden Town, in whom crural phlebitis on the right side had taken place two months before, subsequent to a natural labour. The attack commenced with the usual symptoms of inflammation of the

veins of the uterus. When I visited the patient, who had previously been seen by Mr. Stanley, the affection of the limb was subsiding, but there was still tenderness along the course of the femoral vessels, and the femoral vein could be felt hard and large. In the left inferior extremity, all the branches of the saphena vein were dilated around the knee. On the left side there was no tenderness along the course of the iliac and femoral veins; but there was reason to suspect that the inflammation of the right common iliac had extended into the vena cava, and probably into the left common iliac vein.

Case xxvii.—On the 3d December, 1834, Sir Astley Cooper requested me to see the wife of an old and faithful servant, who was dying after delivery at 71, Portland Street, Portland Town, and who had been attended by Mr. Love, of Brook Street. She was delirious, there was rapid feeble pulse, cold extremities, profuse perspiration, and diarrhœa. The whole of the left lower extremity was swollen but not discoloured, and there was great tenderness along the course of the iliac and femoral veins on the left side. The veins around the knee were unusually turgid.

The usual symptoms of suppurative uterine and crural phlebitis were present, and she died on the 18th, after extensive sloughs had formed over the sacrum. The husband would not permit the body to be examined.

Case xxviii. — On the 12th October, 1835, I saw a patient in St. George's Hospital, under the care of Dr. Seymour, who had been delivered in the Westminster Lying-in Hospital, in February. The labour was natural, but she was attacked soon after with uterine inflammation, for which she was copiously bled, and had warm poultices applied to the hypogastrium. She had remained in a weak state during the summer and autumn; but suckled her child. About the beginning of September, headache and febrile symptoms came on, and pain in the right groin. A week after, the right foot and leg began to swell. The

whole of the right lower extremity is now swollen, hot, and colourless. The foot pits on pressure. Along the leg, thigh, and hip, the superficial veins are greatly dilated. In the right groin, there is a hardness felt, which is painful on pressure. I could not be sure that this was the femoral vein. She had been ruptured on this side. Slight coloured discharge from the vagina. Os uteri healthy. The uterus in its usual situation. Nothing unusual within the pelvis. Pulse 80. Tongue clean. Appetite indifferent. Bowels regular. No cough nor difficulty of breathing, nor pains in the joints.

October 28th.—A few leeches were applied to the right groin, and a tepid lotion with Liq. Ammon. Acetat. to the limb; and the swelling of the limb gradually subsided.

Case xxix.—July 16, 1836, Mrs. D—, æt. 25, Kensington. First child; tedious labour. Now complains of pain and tension in the left iliac fossa, pain on pressure along the inside of the thigh to the knee, then in the ham. Foot and leg swollen, hotter than the other, not pitting on pressure. Not discoloured. Pulse rapid; rigors, sickness at stomach, diarrhœa. Leeches, fomentations, and poultices were applied, and anodynes exhibited internally.

July 17th.—Less pain in the limb, and no increase of swelling. The leeches bled well, and decided relief followed the bleeding.

18th.—Has slept well, and has not complained of acute pain—feels much better. The secretion of milk returning.

25th.—Symptoms declining rapidly. The temperature of the affected limb was ascertained, on the 18th of July, to be 92°, the temperature of the sound limb being 85°. From the 18th to the 24th, the affected limb has been only five degrees higher than the other.

Case xxx.—21st May, 1840, I saw a lady in whom the usual symptoms of crural phlebitis appeared in a mild form, ten days after delivery. There was pain; first appearance

in the left side of the hypogastrium, which gradually extended along the course of the femoral vein. The swelling and tenderness slowly subsided, but though twelve years have elapsed since the attack, the circulation is still carried on imperfectly in the limb.

Case xxxi.—On the 24th July, 1840, I saw a lady in consultation, who had been delivered of her first child, on the 7th July, after a tedious labour, complicated with hæmorrhage. On the tenth day after the confinement, she complained of pain like rheumatism in the right lower extremity. The next day there was swelling of the inside of the thigh, and pain along the course of the femoral vessels. Leeches were applied.

22d.—Pain in the ham, the leg uniformly swollen, hot, and tender along the inner surface. No pain along the outside of the limb.

24th.—The temperature of the affected limb higher than the other. The whole limb swollen, tenderness along the brim of the pelvis or the right side, and along the femoral vein. Pulse 100.

August 8th.—The left lower extremity similarly affected. The swelling and tenderness of the right greatly reduced. The affection of the left lower extremity was accompanied with great constitutional disturbance, and the health was seriously injured.

Case xxxii.—In 1841, I saw a lady in consultation, whose first labour was extremely protracted, and delivery was accomplished by artificial means. Great tenderness of the uterine region followed with fever, and in the course of some weeks crural phlebitis took place in both lower extremities in a mild form. Recovery took place; but in 1845, both limbs were still swollen, and the legs and feet covered with varicose veins.

Case xxxiii.—On the 16th June, 1843, with Mr. Cathrow, I saw Mrs. —, æt. 26, in High Street, Mary-le-

bone, who had been delivered on the 11th September, 1842, of her second child, after a natural labour. Fever took place soon after, with pain in the region of the uterus. Fifteen days after delivery there was pain in the right groin and hip, and the right leg became swollen, and in a few days after the left began to swell, was hot, stiff, and could not be moved. This took place in Northamptonshire, and she has never properly recovered from the attack. Six weeks ago she struck the calf of the leg against a chair, which was followed by pain and swelling. There is now felt a general hardness along the inner surface of the limb, pitting on pressure, the saphena veins are hard and distended, the thigh swollen. Left leg, foot, and thigh hard, and swollen in some parts. Veins distended. No heat of surface. No pain on either side of the hypogastrium. Palpitation of the heart. Tongue clear and moist; appetite good. Catamenia regular. All the means that could be thought of were recommended to restore the free circulation of the blood in the limbs.

Case xxxiv.—On the 19th August, 1843, I saw a patient who had been delivered of her second child, on the 2d February, 1851. The labour was complicated with hæmorrhage, and retained placenta. Symptoms of uterine inflammation occurred in the course of a few days, and this was soon followed by pain in the groins, to which turpentine fomentations were applied. On the tenth day after delivery there were rigors, and the right lower extremity became swollen and painful. Leeches were applied, and mercury given internally. The pain and swelling of the right limb subsided, and the left became similarly affected. Now the left leg has a perfectly natural appearance, the veins are not much distended, and there is no discoloration of the limb; but the patient states that she suffers much from pain about the groins and thighs, and disagreeable nervous sensations about the limb. The right foot and leg are quite well.

Case xxxv.—Mrs. D— was delivered of her first child, after a natural labour, on the 4th September, 1843. No symptom of uterine inflammation succeeded, but during the succeeding three weeks she complained of great weakness, feeling of sinking, and the pulse was rapid.

On the 26th September she complained of pain in the left groin and calf of the left leg, the day after there was stiffness and slight swelling, and tenderness in the course of the femoral vein, and all the superficial veins of the limb were distended. Leeches and fomentations were employed.

On the 29th, the swelling had increased, there was great tenderness in the ham, and along the inside of the thigh; and there was slight pitting of the foot on pressure. Pulse 100.

On the 1st of October, Dr. James Johnson saw the case; but though there was still great tenderness along the course of the femoral vein, he could not be made to admit that the iliac and femoral veins were inflamed or obstructed, or that crural phlebitis was the cause of the symptoms usually observed in phlegmasia dolens, œdema lacteum, depôt laiteux dans la cuissé, or any other term applied to the disease.

On the 12th, the affection was declining, but ever since more or less inconvenience has been experienced from the obstructed state of the veins.

Case xxxvi.—Mrs. C— was delivered in Mount Street, of her first child, on the 12th October, 1843. Ergot of rye had been given towards the termination of the labour, but the placenta was removed with difficulty, and hæmorrhage followed its extraction. At the end of the week she had headache, complained of great weakness and of pain in the calf of the right leg and groin, with stiffness of the whole limb. Leeches and fomentations were applied.

On the 11th, the whole limb was much swollen, and sickness at stomach, and diarrhœa came on. The pulse 125, and feeble. There was great tenderness, not only of the whole hypogastrium, but along the course of the iliac and femoral veins.

This attack of crural phlebitis was severe, but the patient recovered.

CASE XXXVII.—A lady was delivered in Sussex Square, Brighton, after a natural labour, on the 29th August, 1844. Tenderness in the regions of the uterus, severe rigors, and rapid pulse took place in the course of a few days, and gradually the most marked symptoms of uterine phlebitis followed, viz., rigors and perspirations like those of ague, pains and swellings of the joints of the arms and hands, and in the situation of the right parotid gland, bloody expectoration, and dyspnœa, and before death, which took place on the 3d November, phlegmasia dolens in the left lower extremity supervened; and there appeared dark oblong and circular spots on various parts of the body, and extensive sloughing over the sacrum. There were severe nervous symptoms in the progress of the disease.

The body was not allowed to be examined.

CASE XXXVIII.—On the 30th March, 1844, I saw a lady, with Dr. Scott, at Barnes, who had been delivered of her fifth child ten weeks before. A few days after her confinement she was seized with symptoms of uterine phlebitis. In a short time dyspnœa and other symptoms of thoracic inflammation supervened. Three weeks after delivery, the left crural veins became painful, and the whole limb affected with a hot colourless swelling. The fever and pulmonic inflammation continued, and some time after the right lower extremity became similarly affected. The veins of the right leg are now distended, the surface of the limb is of a cream colour, hotter than the other, and does not pit on pressure. There is pain on pressure above Poupart's ligament, on the right side. Sallow complexion. Pulse 120. Left lung dull; respiration short and quick. No expectoration, no sickness, diarrhœa, or rigors.

Blisters were applied to the chest, mild mercurials were exhibited, with anodynes, and slight diet; but death took place not many days after.

Permission could not be obtained to examine the body.

Case xxxix.—On the 27th January, 1845, Dr. Burnie requested me to see a lady who had been delivered of twins, in De Beauvoir Square, about a month before, and who had not recovered favorably. The pulse was 120; there was dyspnœa, with crepitating râle in the chest, and a painful state of the left shoulder-joint with some swelling in the back part of the deltoid muscle. There was no redness, but great tenderness on pressure. There was also great tenderness along the course of the right iliac and femoral veins, and in the situation of the right sacro-sciatic notch. No swelling of the extremity. Pulse rapid and feeble, with diarrhœa.

October 30th.—The whole of the right lower extremity swollen, as in phlegmasia dolens.

The case ultimately terminated fatally.

Case xl.—December 7, 1846, a lady, in Sloane Street, fifteen days after delivery. The affection commenced with pain of the right thigh, iliac nerve, and tenderness of the right groin, and stiffness of the whole limb. Rapid pulse and slight delirium. The whole limb was hot and swollen, and there was slight pitting upon pressure.

The patient recovered.

Case xli.—On the 11th of February, 1845, I saw a lady at Brompton, who had been delivered three weeks before, without any accident. The veins of the right lower extremity had been varicose during pregnancy, and they had become inflamed soon after the confinement. A chain of abscesses had formed along the course of the right saphena vein, which had been opened, and presented a healthy appearance. A deep abscess had formed in the back part of the left hip, and suppuration was going on in the cellular membrane, over the left trochanter. On the 24th, the pulse was rapid and feeble; there was great pain in the right arm, about the elbow-joint, and a large gangrenous slough

had formed over the sacrum, without any of the internal organs having become affected. This patient ultimately died completely exhausted.

The following cases demonstrate that phlegmasia dolens may occur wholly unconnected with pregnancy and parturition, and that in such cases the inflammation likewise commences in the uterine branches of the hypogastric veins, and subsequently extends from thence into the iliac and femoral trunks of the affected side. In some of these cases the inflammation of the uterine veins was produced by cancerous disease of the os and cervix uteri, in others there was no organic disease existing previously of any kind.

Case xlii.—On the 9th May, 1835, I saw a young lady in Bruton Street with Mr. Nicholles, who had been seized soon after her arrival from the Isle of Wight with symptoms of low typhoid fever. For some time before this attack commenced she had been chlorotic, had menstruated sparingly, and had suffered from pain in the back and left hip. Some days before I saw her the whole left inferior extremity had become swollen, tense, and hot, but not very painful. It then looked like alabaster. There was considerable tenderness on pressure along the course of the femoral vein, but the vessel itself could not be distinctly felt. The abdomen was soft and nowhere painful on pressure. The pulse was rapid, the superficial veins distended with blood, the tongue dry and brown.

On the 21st death took place with vomiting and the usual symptoms of low typhus.

On the 22d I was present at the examination of the body. The left common and external and internal iliac veins were thickened, lined with false membranes, and plugged up with coagula of lymph and blood. The internal iliac was most inflamed, and its vaginal and uterine branches contained pus.

Case xliii.—At the beginning of April, 1836, I saw a lady under the care of Dr. Duffin, who had a fibrous polypus

the size of a large pear, with a thick root encircled by the os uteri. Little difficulty was experienced in applying a ligature with the double canula; and the tumour came away five days after in a state of slight decomposition.

On the 26th there were symptoms of inflammation about the left uterine appendages, and tenderness along the crural vessels on the same side. The whole left lower extremity became swollen, as in cases of crural phlebitis in the puerperal state, and the disease after running the usual course, in a mild form, terminated favorably.

Case xliv.—In the month of May, 1831, I saw a woman, æt. 22, in the Middlesex Hospital, who was under the care of Sir Charles Bell. The superficial veins of the left lower extremity were greatly distended, and the thigh, leg, and ankle were swollen and œdematous; there was great tenderness on pressure along the brim of the pelvis, and in the course of the femoral and saphena veins. The right inferior extremity was in the natural state. About six months before, she had experienced a fall in which the lower part of the spine had violently struck the ground; since that time there had been constant pain in the back, with irregular menstruation and prolapsus uteri. Soon after the accident, she also began to suffer from pain along the brim of the pelvis on the left side, extending downwards under Poupart's ligament along the thigh, in the course of the femoral vessels. The veins of the lower extremity in a short time began to enlarge, and also the whole limb became swollen, painful, and œdematous.

Case xlv.—In the autumn of 1831, I saw a young lady in consultation with Mr. Jones, Soho Square, who was suffering from an attack of crural phlebitis of the left side. The whole left inferior extremity was swollen, tense, hot, and painful, but not discoloured. The femoral vein under Poupart's ligament was felt like a large hard cord, and pressure over it and along the course of the iliac veins of the same side produced great suffering. The limb was completely

deprived of the power of moving. The thigh did not pit upon pressure, but the integuments of the leg retained the impression of the finger. The tongue was white, the pulse rapid, and there was great irritability of stomach and depression of strength. This attack was referred to the sudden suppression of the catamenia, from the application of cold and wet, which was followed by great uterine tenderness. The acute symptoms were soon subdued by leeches and warm fomentations to the limb, but the extremity remained weak for some months after.

Case xlvi.—In September, 1830, I saw a lady, æt. 40, under the care of Dr. Copland, who, after being exposed to cold, had a violent attack of inflammation of the bowels, for which copious venesection was required. Great tenderness in the hypogastric region, with pyrexia, continued for some time after, when she began to suffer from pain in the situation of the left crural veins. The whole lower extremity became affected with a hot, tense, and painful swelling, as in puerperal crural phlebitis. The affection had scarcely begun to subside in the left extremity, when she began to experience pain and tension above Poupart's ligament on the right side, and the right thigh and leg also became affected with a hot, painful, colourless intumescence. I saw this lady in consultation with Dr. Copland, when the right extremity had become affected, and the disease did not differ in any respect from the crural phlebitis of lying-in women. By the repeated application of leeches, fomentations, &c., the acute symptoms were soon subdued, but the feet and ankles long remained weak and œdematous.

Case xlvii.—A young woman under the care of Dr. Watson, in 1832, died in the Middlesex Hospital from tubercular disease of the lungs. A swelling of the left lower extremity, similar in all respects to crural phlebitis, had taken place some time before death. The uterus was found to be diseased on the left side. The left common iliac and femoral veins had been inflamed, and the left

internal iliac was converted into a ligamentous cord. It was evident that the inflammation of the veins had originated in the uterus.

Case xlviii.—On the 18th May, 1832, I saw in the Middlesex Hospital a young unmarried woman with varicose veins and extensive swelling of the left lower extremity. The uterine functions had been performed irregularly for many months. There was also great tenderness on pressure in the course of the iliac and femoral veins to the middle of the thigh, and little doubt could be entertained that these veins were inflamed and obstructed.

Case xlix.—On the 31st July, 1833, I saw a woman in the Middlesex Hospital, æt. 39, who had been afflicted for more than a year with cancerous disease of the os and cervix uteri. About the 20th of July she had begun to suffer from great tenderness of the hypogastrium, back and hips, and a few days after acute pain took place in the course of the left femoral vein, which was soon followed by a colourless swelling of the whole limb. There was slight pitting on pressure over the tibia, the superficial veins were greatly distended, the femoral vein was hard and painful about four inches in the upper part of the thigh, the pulse was 130 and small.

Case l.—On the 25th of March, 1830, I examined the body of a woman, æt. 50, who had died of carcinoma uteri, in whom four weeks previous to her decease, the usual symptoms of phlegmasia dolens had appeared in the left lower extremity. There was great tenderness in the course of the femoral vein, and along the inner surface of the thigh and leg to the ankle, and the whole extremity had become tense and swollen. The temperature of the surface was increased, but there was no unusual redness of the skin, and pitting on pressure could only be produced around the ankle, and on the upper surface of the foot.

The upper part of the vagina, os, and cervix uteri were destroyed by cancerous ulceration, and a large opening formed between the vagina and rectum.

The trunk and branches of both internal iliac veins were partially inclosed in masses of indurated cellular and adipose substances, and inflamed through their whole extent. On the right side the inflammation terminated abruptly at the junction of the internal with the common iliac vein. The right common and external iliac veins were healthy.

The left common and external iliac and femoral veins were all plugged up with firm coagula, and lined with false membranes. The vena cava, from the junction of the common iliac veins to the entrance of the vena cava hepatica, was occupied and distended with a soft coagulum, which at the upper part had the appearance of a clot of blood, and did not adhere to the lining membrane of the vein. The inferior half of the vena cava was filled with a firm coagulum of lymph, which closely adhered to the vessel. This coagulum, which was soft and pultaceous in the centre, was continuous with that in the left common iliac vein.

Case li.—On the 15th December, 1830, with Dr. Hugh Ley, I examined the body of Mrs. King, æt. forty-six, who resided at No. 49, Bell-street, Paddington. For upwards of two years she had suffered from great pain in the uterus, sacrum, loins, and thighs, and there had been a copious discharge from the vagina of a serous, purulent, and sanguineous fluid. Her complexion was sallow, and there were frequent attacks of sickness and vomiting. For some time before death, great pain had been experienced when the contents of the rectum and bladder were passing. It was ascertained at an early period, that there was a great, irregular, fungoid mass growing from the orifice of the uterus. In the month of October, 1830, a swelling took place in the calf of the right leg and ankle, which pitted on pressure. The integuments were hot, but not discoloured. On examining the upper part of the thigh, the femoral vein was felt hard, and it was painful on pressure, and there was

also great tenderness in the course of the external iliac vein. The swelling of the limb continued three weeks, and then gradually disappeared. A month after, the left lower extremity became similarly affected, but in a much slighter degree. In both the swelling had entirely disappeared before death, which took place on the 13th December, 1830. Two days after, I inspected the body, and found the fundus uteri natural, but the greater part of the body of the uterus had been converted into a thick mass of a caseous consistence. All traces of the natural structure and the orifice and cervix had disappeared, and they had been changed into a substance like lard. Some portions of this mass were as soft as brain. The right common, internal and external iliac and femoral veins were all impervious, their coats thickened, and filled up with firm coagula of blood. The lower part of the vena cava was lined with a false membrane, which adhered to the inner surface of the vessel. A number of large indurated glands surrounded the lower part of the vena cava, right common external and internal iliac veins. In the centre of some of these glands there was a soft substance like lard, or thick cream.

Cases in which crural phlebitis followed inflammation of the saphena veins, and of the deep veins of the lower extremities, from fracture of the tibia and fibula, and the pressure of encephaloid tumours on the thoracic viscera.

Case lii.—On the 8th February, 1836, I saw a woman, æt. 27, who had been cook in a family for a number of years, and had varicose veins of both lower extremities, and eczema over the legs. The surface was moistened, and nitrate of silver applied, which produced great pain. Severe inflammation of the shin followed, and inflammation of the left saphena vein speedily supervened, and this vessel became hard and painful, as far as Poupart's ligament. In the right lower extremity, the same effect has resulted from the application of the nitrate of silver. Leeches and poultices are now applied to the inflamed veins. The pulse is rapid, the

countenance pale, but there is no other constitutional symptom of phlebitis.

Case liii.—Inflammation of the vena cava, iliac and femoral veins, from fracture of the tibia and fibula. A specimen of fibrinous coagula in the veins in connection with diffuse inflammation of the areolar tissue arising from a compound fracture of the right tibia and fibula, and with large sloughy sores of the integuments of the buttocks, and that covering the trochanters and other prominent parts. The inferior vena cava, to a point about two inches from the liver, and the right common as well as external and internal iliac veins, are seen with some of their smaller branches. They all contained quantities of toughish fibrinous deposits, pretty firmly adherent to the lining surface of the veins,—are greatly thickened, and in places the contained deposit is most intimately united to them. The right external iliac and femoral veins contained a mass of a dark rusty colour, and more easily to be detached from the lining surfaces, which were stained. The vena profunda femoris is seen quite void of any deposit. The light-coloured and somewhat rounded portions of tissue seen united to the lower parts of the preparation, are of thickened and condensed areolar tissue, inseparable from the small venous branches in the cavity of the pelvis. Some of these contained small collections of a puriform fluid. In addition to this firmer deposit, the veins contained quantities of dirty, viscid, chocolate-coloured substance, like softened and broken-down coagula. There was also found extensive peritonitis. The thickened hard membrane lining the pelvic organs binding them down firmly, and the areolar and adipose tissue about the lower part of the bladder and rectum contained numbers of small cavities, with consistent purulent matter within them. This was also the case with the vesical and hæmorrhoidal veins. Secondary lobular pneumonia existed also, and in connection with the fracture of the tibia and fibula there were extensive sloughy sinuses.

The subject of this affection was Edward Miles, a patient

of Mr. Tatum's, who was brought into St. George's Hospital with the above-mentioned fracture of the leg. The wound became unhealthy, and pain in the groin, followed by rigors, came on, and subsequently diffuse inflammation of the areolar tissue of the entire limb, and a gangrenous state of the foot. Bed-sores, and repeated attacks of the inflammation came on, and he sank and died July 11, 1851.

CASE LIV.—Inflammation and obstruction of the lower portion of the vena cava, and the right iliac and femoral veins, produced by an encephaloid tumour at the upper part of the chest.

The right lower extremity was enlarged throughout, the superficial veins were distended. There was a tumour of tolerable firmness, the size of an orange, at the upper part of the chest and a little to the right of the upper bone of the sternum. There was also a large tumour, of about the size of two fists, to the right of, and under the right mamma. In both cases the integuments were entirely healthy. The large tumour pressed into the thoracic cavity, united by adhesions intimately with the lungs and pectoral muscles. These tumours presented the same characters, and were examples of encephaloid cancer. The lungs were healthy. The contents of the abdomen were healthy, except the lower part of the vena cava and the right iliac and femoral veins, with their branches, which quantities of thick coagula firmly adherent to the parietes, and going on in parts to softening and conversion into a brownish thin rusty fluid.

This man was 60 years of age. His name was James Lang, and he was under the care of Dr. Wilson, in St. George's Hospital.

CASE LV.—On the 28th September, 1848, I saw a patient, in the Royal Mews, Pimlico, with Mr. Du Pasquier, who died from inflammation of the saphena veins of the right leg, who had been delivered, a few days before, of her first child after a tedious labour. The veins of both lower extremities had been varicose during pregnancy, especially the right. Two days after delivery, she complained of

stiffness of the left leg above the ankle-joint. The part above the ankle on the inner surface was red and swollen, and the branches of the saphena vein up to the ham hard and painful. The pulse was 130. On the following day she was very restless; there was great swelling of the leg, and great tenderness along the course of the saphena vein in the thigh (pulse 140) as high as the groin; a few leeches were applied; much irritation; there was sickness at stomach; great prostration of strength; tongue furred. Died the same night.

The body was examined by Mr. Du Pasquier, and the following is his report of the appearances:—"I examined the veins of Mrs. G—'s leg last evening, it was filled the whole length, from the ankle to the groin, with dark coagulated blood, which stained the cellular tissue which surrounded it, with the same colour. There was no appearance whatever of pus either in the vein itself or in the cellular tissue, at least I could not detect it."

The elbow-joint was swollen and puffy, and the veins around it of a dark colour as in the leg.

Case lvi.—Mrs. N—, æt. 37, 1st of July, 1830. Three months ago a small ulcer appeared above the left internal malleolus, with much inflammation of the surrounding integuments. A varicose state of the veins of the leg had existed some time before. The ulcerations were healed in three weeks, but the saphena veins along the inner surface of the leg, knee, and thigh to the groin, became hard, and exquisitely painful. This painful condition of the veins has been gradually increasing, and a general hot and colourless intumescence of the whole limb has taken place. The veins around the ankle can now be felt, indurated and knotted, and in three joints along the front of the tibia, there is a circumscribed hardness, with intense redness of the integuments. There is exquisite pain on pressure along the whole course of the saphena vein in the thigh. The femoral vein, three or four inches under Poupart's ligament, is hard and painful, and pain is experienced on pressure

along the brim of the pelvis. The hypogastrium, more particularly on the left side, is tense and swollen, and she complains of a distressing sense of pulsation or throbbing in the lower part of the abdomen. For several days there has been retention of urine; the countenance is anxious and depressed; there are tremors of the muscles of the face and extremities; tongue furred; occasional retching; urgent thirst; respiration hurried; slight cough; pulse 120.

4th.—The limb is less swollen, but there is still great tenderness in the left side of the hypogastrium, and along the inner surface of the limb. Constitutional symptoms somewhat relieved. Great prostration of strength.

27th.—Leeches, &c., have been repeatedly applied along the course of the affected vessels, and the tenderness is now much relieved. Sickness, with foul tongue, and quick pulse, continue.

14th of May, 1830.—Health improved. There is considerable enlargement of the affected extremity, and there are large clusters of purple veins around the ankle. There is now a hard tumour of considerable size in the situation of the left ovarium, and she has lately suffered much from prolapsus uteri, and uterine irritation, with leucorrhœa; she menstruates regularly; the right lower extremity natural.

18th of October, 1831.—There is much hardness and tenderness on pressure in the situation of the left femoral vein. The extremity is still swollen, of a deep purple colour, and the foot and ankle covered with enlarged veins. The abdomen is swollen, but no fluctuation is perceptible.

The cases and dissections of the distinguished pathologists previously referred to, and those contained in this and my other communications to the Society on Phlegmasia Dolens, prove in the most conclusive manner that inflammation of the iliac and femoral veins, is the proximate cause of the disease; and that in puerperal women the inflammation commences in the uterine branches of the hypogastric veins. It has likewise been demonstrated by morbid anatomy, that phlegmasia dolens is a disease which may take place in

women who have never been pregnant, and in the male sex, and that under all circumstances the proximate cause is the same. Respecting the treatment of phlegmasia dolens, considerable diversity of opinion has prevailed. Mauriceau, who first described the disease, was aware of its danger when the swelling of the limb was very great and painful, accompanied with symptoms of inflammation; suppression of the lochia; fever; difficulty of respiration; and great tension of the hypogastrium; but for such cases he has not described any method of treatment. In the milder forms of the disease he chiefly trusted to diuretics and purgatives, and has made no allusion to bloodletting or local treatment.

Puzos, who published three memoirs, 'Sur les Depots Laiteux, appellés communément Lait Repandu,' in 1759, and who described not merely the local but the constitutional symptoms of uterine and crural phlebitis with the greatest accuracy, employed repeated bloodletting from the arm for the hypothetical purpose of evacuating from the blood the milk formed in it during pregnancy and in the puerperal state, and which imaginary milk, he says, "*roule confusément avec le sang, dans toute l'habitude du corps.*" Purgatives, diaphoretics, and hot fomentations, cataplasms, and frictions with volatile liniments, were the remedies he chiefly employed after the repeated abstraction of blood from the arm. Although Puzos was convinced of the necessity of venesection in those *depôts*, accompanied with fever and which at the commencement were acute and inflammatory diseases, he acknowledges that the remedy was not infallible, and that he had lost several patients, "malgrè toutes les saigneés que j'avois pû faire."

"In every case, however," says Dr. Davis, "where I have been a party to the adoption of such practice, or where I have had the opportunity of observing it in the practice of others, I feel it my duty to state that it has completely disappointed expectation." Dr. Davis recommended the local abstraction of blood by leeches, blisters, evaporating lotions, free and constant exposure to the action of the atmosphere, and the internal exhibition of digitalis and blue pill.

In all the cases of phlegmasia dolens which I have witnessed, the pulse has been so rapid and feeble, and there has been so much constitutional debility, as usually observed in uterine phlebitis, that I have not ventured to recommend general bloodletting. There are cases, however, which have been observed by others, in which the abstraction of a moderate quantity of blood from the arm has appeared at once to break the force of the attack. In a great proportion of cases venesection is not required, and we may trust for the relief of the inflammation to the repeated application of leeches above and below Poupart's ligament in the course of the iliac and femoral veins. Two dozen of leeches should be applied immediately after the commencement of the disease, and the bleeding should be encouraged by warm fomentations and warm linseed meal poultices. Should the relief of the pain not be considerable, it is requisite to reapply the leeches in numbers proportioned to the severity of the attack, and to repeat them a third, or even a fourth time, at no very distant intervals, should the disease not yield.

Some patients have derived the greatest relief from the use of warm cataplasms to the limb, while others have derived most advantage from tepid evaporating lotions.

The bowels are often much disordered in this disease, but the employment of acrid cathartics is injurious. Repeated small doses of calomel and antimonial powder should be given, with some mild purgatives, not only with the view of correcting the deranged state of the digestive organs, but to subdue the local inflammation and the great constitutional disturbance usually present. It is of importance also to administer saline and diaphoretic medicines, and to procure rest, and relief from pain by anodynes, until the acute symptoms pass away. The diet should be the same as that usually allowed to patients who are labouring under inflammatory and febrile diseases. I have seen no advantage derived from the use of digitalis in any stage, either of uterine or crural phlebitis.

When the acute inflammatory symptoms have passed

away, the limb remains in a weak and œdematous state, and great uneasiness is often experienced from congestion of blood in the veins. Until the collateral branches which are to carry back the blood to the head become enlarged, it is impossible by any means to afford complete relief. Much benefit may, however, be derived in this stage of the complaint from the application of leeches to different parts of the limb, where there is much tenderness, and by piercing it in the horizontal position. I have seen mischief produced by having recourse too early to remedies intended to promote the absorption of the fluid effused into the cellular membrane. Blisters, friction, stimulants, embrocations, and bandages to the limb, are only useful when the inflammation of the veins has subsided, and other vessels have become so much enlarged as to carry on the circulation of the blood in the extremity without interruption.

I have not perceived any sensible benefit accrue from the use of mercurial ointment and iodine in phlegmasia dolens, and I consider the local abstraction of blood at the commencement of the disease to constitute the most important part of the treatment.

POSTSCRIPT; *September*, 1853.

CASE XXXV.—This lady died from tubercular phthisis, on the 16th Sept. 1853, and on the 18th George Pollock, Esq. examined the body. He removed the iliac and femoral veins of the left side, and they are now in my possession. Bands of false membrane are seen extending across both the iliac and femoral veins, and their canal is greatly contracted but not wholly obliterated. All the structures surrounding the veins appeared healthy.

ON

THE USE OF TWO NEEDLES AT ONCE

IN CERTAIN

OPERATIONS ON THE EYE,

ESPECIALLY IN THOSE FOR

CAPSULAR CATARACT AND ARTIFICIAL PUPIL.

BY

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Received April 12th.—Read May 24th, 1853.

I am desirous of describing a new mode of operating applicable to many cases of closed or obstructed pupil, and which will be found, I think, a useful addition to the resources of ophthalmic surgery.

It consists in the simultaneous employment of two needles, introduced at different points through the outer coat, and made to act in concert upon false membranes, opaque capsule, or iris, or even on the lens itself under certain circumstances.

Several advantages attend this mode of operating. Opaque portions of capsule are often very tough, and being attached to the suspensory ligament of the lens, or to the pupillary border of the iris, these extensile structures readily allow the opaque membrane to recede before the needle, rather than be torn or cut through, and the surgeon vainly sweeps the membrane before the instrument from side to side at the

risk of serious injury and consecutive inflammation of the ciliary processes or iris.

Now if two needles are brought to bear on the opaque capsule from different sides of the cornea or sclerotica, each furnishes the other with a point of resistance, and the capsule may be torn open or cut at pleasure, in almost any direction, and generally without any drag on the neighbouring vascular structures.

If the capsule is membraniform, the needles are made to puncture it close together from opposite sides, and their points are then separated so as to tear it open. If it is of a cobweb form they enter one of its meshes, and are used in the same way. If it is a tight band stretched across the pupil, the needles cannot always be so successfully employed, at least, unless the surgeon has acquired some dexterity in handling them. It is then possible to twist one of them round and round the band, and so to get a kind of hold upon it, in case it should on trial have proved too tough to yield to the needles made to cross over it from opposite sides. In one instance only have I been foiled in the attempt to divide such a band with the two needles.

The needles usually act perfectly if passed through any convenient opposite points of the margin of the cornea, the pupil being always, where possible, dilated by atropine. The injury inflicted on the organ by this mode of procedure is thus reduced to a minimum, the simple needle-punctures through the cornea being in themselves trivial, and no other structure being interfered with except the capsule or membrane, which it is the object of the operation to deal with. It is but seldom desirable, for the sake of the manipulations, that one of the needles should be entered through the sclerotica. Of course, in thus using two needles at once, in knife-and-fork fashion, the operator's hands are both engaged, and the lids must be held open for him, either by an assistant, or, what I much prefer, by the wire speculum. This leaves the globe completely at his command, with room for all the movements of the needles.

A great advantage of this mode of operating is, that it

may be at once proceeded to in any case, where, on trial, a single needle is found to be insufficient to effect the laceration of an obstructing membrane. Sometimes, and indeed generally, the toughness of an opaque capsule is obvious to the experienced eye; but at others, the needle alone can afford a correct intimation of its texture; and if, where the surgeon suspects that he may meet with resistance, he begins by using the wire speculum, and provides two needles in case of need, he can make trial with one of them first in the ordinary way, and proceed to the operation now described, by inserting the second needle should he find it necessary to do so. He is thus able exactly to adapt the extent of his interference to the demand of each particular case, and this without deferring the major operation to a future time.

The needles to be used should be ordinary cataract-needles, the stem cylindrical, and of a size to easily occupy the corneal puncture, and allow of free movement while retaining the aqueous humour. If the stem does not move freely in the puncture, the cornea is apt to be *creased*, and the view of the interior of the eye to be thereby obscured. They may be made to cut more or less near the point. It is well in some cases, that one or both should be slightly curved at the point. It is better to insert one first down to the membrane, this holds the globe, and the surgeon sees more deliberately what he is doing. The second may then be carefully introduced so as to act in the way previously determined on in the operator's mind; for he should consider in what direction the membrane may be best torn through in each case before commencing, since on this will depend his choice of the points at which to puncture the cornea or sclerotica. In general, it is better to hold one needle fixed while the other is moved, as it is not easy, without practice, to attend accurately to the movements of both at once. The temper of the needles should of course be such that if they chance to touch, there may be no risk of their points breaking. To avoid the chance of inadvertently penetrating further than desirable with one of them, whilst the attention is engaged upon the other, the stem of

the needles may be made thicker from the handle up to within half an inch of the point ("stop-needle"); in no case is it necessary to enter the instrument beyond half an inch in depth.

The patient should be laid on his back, and chloroform may be exhibited if he thinks it worth while.

In the preceding remarks, I have had chiefly in view the application of the double-needle-operation to tough capsular and membranous cataracts. Many surgeons of great experience are averse from meddling with such cases if they can possibly avoid it, that is, if the patient already enjoys a moderate amount of sight, or if the opposite eye is sound. They are deterred by the fear of exciting dangerous, or, at least, troublesome inflammation, by the injury liable to be done to the iris, ciliary processes, or even (through the vitreous humour) to the retina, in the attempt to tear through the opaque capsule. But I believe that by operating in the way I propose, this danger may be almost entirely avoided, by the power it confers of strictly limiting the injury to the punctures of the cornea and the capsule itself.

The operation is also suitable to cases of false membrane obstructing the pupil where the iris is adherent to the lens, and where it may seem desirable to get rid of the lens by solution. In several instances of closed pupil I have employed, in the first instance, Mr. Tyrrell's operation of drilling, so as to destroy the body of the lens, and afterwards, when a dense membrane alone remained, I have used the two needles to tear open this membrane and restore the pupil in its natural situation. To do this with a single needle is often exceedingly difficult, owing to the toughness of the membrane, and the extensibility, or even the soft and rotten state of the iris, altered in texture by inflammation. A single needle passed through the pupil may often be pushed widely from side to side, under such circumstances, without dividing the false membrane, and sometimes the iris has been separated from the ciliary attachment in the effort, or such inflammation has followed its stretching, as to close again the pupil which may have been opened, and thus to nullify the whole proceeding. A second needle in

the pupil affords the requisite point of resistance, and the slightest force will now suffice to establish the aperture, without the least strain upon the tissue of the iris.

I have further made use of the double-needle-operation to form an artificial pupil in some cases, chiefly those following extraction of cataract, or where the lens, from any cause, has been removed. With two cutting needles inserted into the iris at the same point, and then diverged, a new pupil may be produced with great precision, and with accurate regard both to size and situation: it may be kept of moderate dimensions, and be placed in the most central position possible,—conditions of which I have elsewhere endeavoured to signalise the importance.[1]

I might adduce many examples in illustration of the above observations. The method I advocate has been extensively tested during the last six months by several of my colleagues, at the Moorfields Hospital, as well as by myself, both there and in private practice; and it has been frequently remarked in the operating theatre, by those who have witnessed the operations performed, that results have been obtained by means of it which could not have been otherwise arrived at. Very frequently have two needles been able to effect the division or removal of false membranes, where one only had a moment before proved unavailing. I therefore feel justified in speaking of this method as one enabling the surgeon to deal with the important morbid states in question more effectively as well as more safely than has hitherto been possible.

I will conclude this brief paper with a few remarks on the canula-instruments recently introduced for operations on opaque capsules, and which the double-needle-operation is calculated, in my opinion, in most of such cases to supersede, though these instruments will still be highly available in certain operations for artificial pupil. All must admire the delicacy and beauty of the canula-forceps and the canula-scissors, in their several modifications of self-penetrating, blunt-pointed, &c. With great nicety in their construction

[1] Medical Times, Jan., 1852.

and management, and with a little practice, they may be manipulated so as to effect all that has been expected of them. But the canula-forceps act by forcibly dragging the dense and tough capsule from its attachments, and of course they must endanger the vascular parts to which the capsule is connected, in precise proportion to the firmness of these attachments; and though the immediate result, in the clearing of the pupil, is admirable, and often followed by but trivial inflammation, yet violent and even destructive ophthalmitis occasionally ensues, particularly where the eye has previously suffered, and where it is usually of most essential importance to avoid re-exciting it. As respects the removal of the fragments of capsule out of the organ, no advantage attends it, except the security it gives that they cannot again obstruct the pupil; but fresh inflammation, induced by the removal, may lead to a new membranous cataract, and, on the other hand, portions of capsule divided by the double-needle-operation, and receding behind the pupil, are as much out of the course of the light as if they were actually withdrawn from the globe, and they can occasion no irritation.

The canula-scissors of my friend, Mr. Wilde, inflict less injury on the vascular parts than the canula-forceps, because they cut across the capsule without dragging on its attachments; but they require room for the blades to work in, and it is therefore essential that the aqueous humour should be retained, which is not always easy, unless they are introduced through the sclerotica. They require a largish orifice to enter by, and they cannot readily cut the capsule except in the direction in which they have been introduced, so that they seem chiefly applicable to cases in which a single and very tough band stretches across the pupil. To divide this, I believe they are still the best instruments.

ANALYSIS OF THE CASES

OF

INJURIES OF THE HEAD,

EXAMINED AFTER DEATH IN ST. GEORGE'S HOSPITAL,

From January, 1841, *to January*, 1851,

WITH

PATHOLOGICAL AND SURGICAL OBSERVATIONS.

BY

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Received April 13th.—Read May 24th, 1853.

Thinking that some important points of practice might be illustrated by the classification of a large number of fatal cases of Injuries of the Head, occurring in a civil hospital, I venture to lay before the Society an analysis of all those cases, which were examined after death at St. George's Hospital, during a period of ten years. In this analysis, I have purposely included only those cases which were examined; as it is in fatal cases alone that the exact nature of the injury, and its extent, can be clearly made out. The observations are confined to cases which have occurred at St. George's Hospital; and those points, the value of which has been tested, either in the wards, or in the dead-house of the hospital, have been principally dwelt upon.

The present Paper relates simply to Scalp-wounds, and to Fractures of the Skull; but it is my intention, should this communication be favorably received, to bring before the Society, at an early period, the analysis of the other injuries belonging to this subject.

Section I.

Scalp-wounds, without Fracture of the Bones.

In this decennium, 33 cases of scalp-wounds, without fracture of the bones, were examined. In 10 of these cases, death was produced by some other cause, there being no unhealthy appearance, either about the wounds, or the bones of the cranium. In the remaining 23 cases, mischief of a serious nature soon followed the injury, and ultimately proved fatal. In these 23 cases, the wound in 7, was on the forehead; in 7, it was at the back of the head; in 6, it was in the parietal region; in 2, it was in the temporal region; and in 1, it was on the crown of the head. In 8 cases, the wound was large, extending, in 2 cases, the whole length of the skull, from the front to the back part; in 9, the wound was contused; in 7, the surface of the pericranium alone was exposed; and, in the remaining 16, the bone was either laid bare at the time of the accident, or became so shortly afterwards.

Diffuse cellular inflammation occurred in 17 cases, and in 12, this was accompanied by erysipelas. This inflammation presented itself under the occipito-frontalis muscle, in the shape of simple œdema, of lymph and pus, or of suppuration and sloughing, undermining, and, in one instance, completely separating the scalp from the parts below. In 4 cases, there was also diffuse inflammation of the neck, which spread down to the mediastina in 2 cases, and it was accompanied by œdema of the larynx in 2. The diffuse inflammation, running on to sloughing, was in 2 cases followed by hæmorrhage, which in 1 was of a very severe character.

In 12 cases, no traces whatsoever of inflammation were found, either in the membranes or in the brain; of these, the diffuse inflammation proved fatal in 3; and in the other 9, death was caused by purulent infection: in addition to the injury of the head, there was in 1 of these simple fracture of the internal malleolus, and in 1 there was fracture of several ribs.

In 10 of the remaining 11 cases, inflammation existed about the membranes or the brain, in 8 of which suppuration, more or less extensive, was found between the bone and the dura-mater; 5 of these were complicated with purulent infection. In addition to the injury of the head, there was also in 1, compound fracture of the leg; and in 1, fracture of the spine, but this was not accompanied by any symptoms.

In 1 case, sloughing of the dura-mater, without any injury to the internal part of the bone, had taken place opposite to a large abscess, which originally had begun in the structure of the brain.

In all these cases, the bone was exposed; in 4, the exposed bone was discoloured, being of a dark, or of a yellow tinge; in 5, matter was detected in the diploë. The trephine had been applied in 3 of the cases of suppuration between the bone and the dura-mater, as well as in that of the abscess of the brain.

These 23 patients were, for the most part, persons of dissolute habits, and many were addicted to hard drinking. Several of them had insisted on leaving the hospital, but were readmitted on account of the diffuse inflammation which had subsequently made its appearance.

The diffuse inflammation proved fatal in 3 cases: in 1 patient, æt. 32, the inflammation was confined to the soft parts about the skull,—he lived seven days: in 1, æt. 65, who lived nine days, the inflammation was very extensive, and accompanied by erysipelas, which spread over the face: in the third case, there was also diffuse inflammation of the neck, spreading down to the mediastina, with œdema of the chordæ vocales; the patient, æt. 65, lived eight days.

In hospital practice, an injury of the scalp, even of a slight nature, is not unfrequently followed by an œdematous swelling, which may spread over the whole scalp, and in such cases, simple acupunctures, if made at an early period, and repeated according to circumstances, as pointed out by Sir B. Brodie, are generally sufficient to prevent any further mischief. Should the inflammation run on to the effusion

of lymph or pus, then free incisions become necessary. Sometimes, notwithstanding these free incisions, now commonly made in diffuse inflammation of the scalp, the cellular tissue sloughs; but, generally, if this treatment has been carried out at an early period of the attack, and to the necessary extent, the skin may be saved. In a few cases, the sloughing process may extend over the whole surface of the skull, from the front to the back part, and, after the separation of the sloughs, the over-distended and perfectly loose scalp, presenting a puckered and baggy appearance, looks by far too large for the cranium, so large that it might be thought impossible for the parts ever to regain their natural size; and yet, after a time, the skin is observed to be less and less flaccid, and ultimately it readapts itself most perfectly. Should the skin and the pericranium, fortunately a very uncommon occurrence in the present day, also become involved in the sloughing process, the bones of the skull may become exposed to a very great extent. A middle-aged woman, who was admitted into St. George's Hospital, under the late Mr. Walker, with a burn occupying the neck, and other parts of the body, was attacked with diffuse inflammation of the scalp, which, notwithstanding the incisions which were made, ended in extensive sloughing of the skin, with separation of the pericranium, and denudation of the bone, involving the frontal, both parietals, and a large part of the occipital, after which the patient lived some time, and ultimately died exhausted.

This sloughing may be followed by hæmorrhage, sometimes of a serious nature, the source of which may be a matter of great uncertainty. Should the sloughing be confined to one part, the source of the bleeding may be easily ascertained; but, should the bleeding occur in one of those cases where the whole scalp has become detached, it will lead to great doubt and perplexity. Being in the wards of the hospital, I was once sent for to a patient suffering from a sharp attack of hæmorrhage, in whom the whole scalp had been detached by extensive sloughing of the cellular tissue, following diffuse inflammation. I found the scalp, previously

loose and baggy, now stretched, and made tense by a large accumulation of blood under it, the more fluid part of which was pouring out in various directions through several large incisions made some days previously. It was evident that this hæmorrhage was proceeding from some large vessel, but it was difficult to say whether it came from either of the temporal arteries, or even the occipital arteries. Having established pressure on the trunks of these four vessels the blood was pressed from under the scalp; the pressure was then taken off from the vessels one by one, and the bleeding was found to arise from one of the occipitals, or some of its large branches. As pressure controlled the hæmorrhage, a pledget of lint was applied over the artery, low down on the occipital bone, and firmly fixed there; this had the desired effect, and no more bleeding occurred.

Separation of the dura-mater from the bone may occur either as a primary, or as a secondary effect. In the first, the small vessels connecting the dura-mater to the bone are ruptured by the blow, and the membrane is in consequence detached from the bone to a greater or less extent. In the second, the osseous tissue itself inflames and suppurates, either in consequence of the contusion of its diploë, or in consequence of the bone being deprived of its periosteum.

Generally speaking, the suppuration between the bone and the dura-mater is circumscribed, and the extent of the mischief on the inner side of the bone is exactly traced by that on the outer side; but, should the suppuration occur in the parietal region, the mischief may be of a much more diffuse character, and that too without any evidence of its extent on the external parts. Occurring in the parietal region, the matter may follow the course of the branches of the middle meningeal artery, and running along the cellular tissue surrounding these vessels, it may reach the base of the skull, and extend as far as the foramen spinosum. Of the eight cases of suppuration between the bone and the dura-mater, observed in these ten years, three presented this form of the disease.

In this decennium, there has been no single instance, nor

indeed do I ever recollect having seen one, of the secondary puffy tumour of the scalp, described by Pott and others, as indicative of mischief between the bone and the dura-mater.

Suppuration between the bone and the dura-mater appears not only to have been much more frequent, but also very much more commonly confined altogether to this situation formerly than it is in the present day.

In the second section of 'Injuries of the Head,' Pott gives twelve cases of scalp-wound followed by subsequent mischief beneath the bone, in seven of which the suppuration was altogether confined to the outer surface of the dura-mater. In the various cases which I have noticed of this affection, I have never seen a single instance in which the suppurative inflammation was thus limited; in every case which I have examined, inflammation has at the same time existed beneath the dura-mater. It cannot be said that this very marked difference depends upon the amount of inflammation, or the quantity of matter thrown out, for in several of Pott's cases not only was the quantity of matter thus situated very large, so large indeed that it was thought impossible for the patients to recover, but the inflammatory process had been going on for some days, as shown by the symptoms and the quantity of matter evacuated from the spot where the trephine was applied; whereas, in several of the cases which I have noticed, although the extent of the mischief between the bone and the dura-mater has been comparatively trifling, still the diffuse inflammation beneath the membrane has been most extensive. In every one of the eight cases of suppuration between the bone and the dura-mater occurring in this decennium, puriform effusion was found within the cavity of the arachnoid, and appeared to have spread from the external to the internal parts. That this difference does not depend upon locality is evinced by the results of similar cases in different places and in different countries.

Suppuration between the bone and the dura-mater naturally leads to a practical question of the utmost importance, I mean the application of the trephine. Formerly,

and especially in Pott's hands, it appears in such cases to have been one of the most successful operations. Mischief was suspected under the bone, the trephine was applied, matter was found, in some cases in large quantities, and evacuated, and the patients recovered without any further difficulty. Such is the history of several of Pott's cases; but, unfortunately, we have met with no such success. It has never yet fallen to my lot to see a single instance in which the application of the trephine has, under such circumstances, had a successful issue. In every case in which I have seen the operation performed, the patient, notwithstanding the evacuation of the matter, has died of diffuse inflammation of the membranes. Still, notwithstanding that our efforts have been so unsuccessful, we do not hesitate, whenever the various symptoms lead to the diagnosis of matter between the bone and the dura-mater, to apply the trephine, and we give the patient the only chance, however small that may be, of recovery; but, on the other hand, we consider the application of the trephine altogether useless in cases of intra-cranial suppuration, where the symptoms are those of acute inflammation of a diffuse kind, in which the signs of compression are generally but slightly marked.

Of the eight cases of suppuration between the bone and the dura-mater without fracture, noted in this decennium, five were not operated upon, and three were trephined. In the first class, in which no operation was performed, the symptoms of compression were either but very slightly marked, or altogether absent.

A man, æt. 20, was admitted into St. George's Hospital, under the care of Mr. Cutler, in July 1845, with concussion of the brain, and a small scalp-wound, exposing the right parietal bone. He went on well for a fortnight, but symptoms of inflammation of the membranes then began to show themselves; he was bled, and put upon mercury, but the symptoms became more marked, and profuse perspirations and severe rigors soon made their appearance; he

lived twenty-nine days after the accident, there never having been any symptom of compression, save a slight drooping of the right eyelid. On removing the skull cap a small quantity of lymph and pus was found under the exposed bone, which itself was of a dark yellow colour; from this point lymph and puriform fluid were traced along the cellular tissue of the branches of the middle meningeal artery to the base of the skull, where suppuration also existed around the various foramina of the sphenoid, as well as in both orbits. In all these parts, the dura-mater was most readily detached from the bone. There was also extensive effusion in the cavity of the arachnoid, and in the sub-arachnoidean tissues, with inflammation of the brain. Both lungs presented marks of purulent infection. The other viscera were healthy, excepting the kidneys, which were mottled.

A man, æt. 50, who was admitted into St. George's Hospital, under the care of Mr. Cæsar Hawkins, with an extensive and bruised wound of the scalp, not exposing the bone, was attacked with diffuse inflammation and sloughing, which ended in exposure of the parietal bones to some extent; he was apparently recovering from this attack when severe symptoms of diffuse inflammation of the membranes made their appearance. Two days before his death he had a rigor, which was followed by spasm of the right arm, then paralysis of the right leg, and at last he became perfectly insensible. He lived eighteen days; the most prominent and earliest symptoms having been those of inflammation of the membranes of a diffuse character. The skull cap having been removed, nothing was observed under the exposed part of the right parietal, but a small quantity of pus was found immediately under the exposed part of the left parietal, the diploë of which was extensively infiltrated with matter; pus was also traced some distance along the middle meningeal artery, and in the structure of the dura mater. Large quantities of lymph were found both in the cavity of the arachnoid and in the sub-arachnoidean tissues,

but the effusion was principally confined to the upper surface of the left hemisphere. Some puriform fluid existed in the superior longitudinal sinus.

A man, æt. 30, who was admitted into St. George's Hospital, under the care of Mr. Tatum, with a large scalp-wound, exposing a portion of the left parietal, was attacked with diffuse inflammation of the scalp, and symptoms of inflammation of the membranes; he lived sixteen days, during which time he never had any symptoms of paralysis. Immediately under the denuded bone there was a small quantity of pus; puriform effusion was also traced in the cellular tissue, along the course of the middle meningeal artery, and in the dura-mater. The cavity of the arachnoid and the sub-arachnoidean tissues, on the left side, were filled with lymph and pus: the corresponding portion of the brain was inflamed.

A man, æt 60, was admitted into St. George's Hospital, under the care of Mr. Tatum, in June, 1847, for a large scalp-wound not exposing the bone; he went on well for seven days, when diffuse inflammation made its appearance about the scalp; sloughing of the cellular tissue ensued, and the bone became exposed. He recovered from this attack, and progressed favorably for some time, when he was seized with inflammation of the membranes of the brain. He lived five weeks, and never had any signs of compression. Under the exposed bone, which was of the size of a shilling, was a small quantity of purulent lymph on the dura-mater; the bone itself was discoloured throughout and dark; large quantities of sero-purulent fluid, with a thick coating of lymph, were found both in the cavity of the arachnoid and in the sub-arachnoidean tissues; the brain was inflamed; lymph and pus existed in the superior longitudinal sinus, and the right lung was affected with purulent infection.

A very stout, middle-aged man was admitted into St. George's Hospital, under the care of the late Mr. Walker,

in November, 1842, with a scalp-wound in the right temple, laying bare the bone; diffuse inflammation of the scalp having supervened, incisions were made, but a portion of the occipital subsequently became exposed; he lived one month. Under the exposed bone, in the right temple, which was of a scabrous appearance, the dura-mater was inflamed, thickened, and partially separated; and, exactly limited to this spot, there was an effusion of lymph and pus on the parietal layer of the arachnoid, but all the other parts of the membranes and of the brain were quite healthy. Nothing was observed under the exposed portion of the occipital. There was recent effusion in the right pleura, with secondary deposits in both lungs.

Whenever I have seen the trephine applied at St. George's Hospital, for intra-cranial suppuration, in addition to other symptoms, those of compression have been among the most prominent, and the operation was performed under the supposition that the matter would be found between the bone and the dura-mater.

A lad, æt. 16, was admitted into St. George's Hospital, under the care of Mr. Cæsar Hawkins, in August, 1848, with slight concussion of the brain, and a small scalp-wound exposing the bone, which had been caused by a fall from a horse. He went on well for nine days, when he was attacked with feverish symptoms and pain in the head, which were relieved by bleeding and purgatives. On the twelfth day, there was vomiting, with a rigor and profuse perspiration, but there was no pain in the head; on the thirteenth day, the wound presented an unhealthy appearance, the bone was quite dry, the right eye-lid drooped, and the pupil was sluggish, and there was loss of power on the left side, but sensation still remained. One crown of a trephine was applied over the denuded bone, and a small quantity of matter was evacuated from between the bone and the dura-mater; some matter also appeared to come from the diploë itself. The relief was but partial, and the

patient died comatose on the following day. The diploë of the frontal bone, in the neighbourhood of the spot where the trephine had been applied, was infiltrated with pus and very offensive. The dura-mater was separated from the bone to the extent of the size of a shilling, and the cavity from which the matter had been evacuated was strictly limited to this spot. There was extensive effusion of puriform fluid in the cavity of the arachnoid, and in the sub-arachnoid tissues; the corresponding portions of the brain were of a slate colour, soft and pulpy. The lungs and the liver were quite healthy.

A man, æt. 45, was admitted into St. George's Hospital, under the care of Mr. Keate, in May, 1845, with a large scalp-wound, extending nearly the whole length of the right side of the head, and partially exposing the bone. Everything went on favorably for thirteen days, but the wound then put on an unhealthy aspect, and the patient appeared drowsy; he made no complaint, however, and the pulse was natural. On the seventeenth day, there was a slight rigor, which was thought to depend upon some matter confined to the scalp; this was let out, but, on the following day, there was a fit, accompanied by another rigor, and the right side of the body became paralysed; there was also occasional wandering, and at other times the mind was apparently not affected. Two portions of the parietal appearing of a dark green colour, the trephine was applied over them; some yellow lymph was found on the surface of the dura-mater, and the bone itself evidently contained matter in its diploë. The cerebral symptoms went on increasing, and the patient died on the third day after the operation. In addition to the mischief on the outer side of the dura-mater, there was extensive effusion of puriform fluid in the cavity of the arachnoid, and in the sub-arachnoid tissues. Pus was found in the diploë, and in the superior longitudinal sinus. There was also purulent infection of one lung, with pleurisy and pneumonia.

A man, æt. 25, was admitted into St. George's Hospital,

under the care of Mr. Cæsar Hawkins, in November, 1849, with slight concussion, and two large scalp-wounds, one of which exposed the bone on the vertex. The wounds were followed by erysipelas of the scalp, from which he recovered; but, on the tenth day, there was a rigor, with slight delirium and great prostration. On the twelfth day, partial paralysis of the left arm and leg supervened; this became complete on the following day, when there was also loss of power over the rectum and the bladder. The bone corresponding to the wound on the vertex was quite dry. Two crowns of a trephine were applied here, and half an ounce of fetid pus and grumous blood was evacuated. No relief followed the operation, and the patient died on the following day. In addition to the mischief between the bone and the dura-mater, there was an extensive effusion of puriform fluid in the cavity of the arachnoid on both sides, as well as in the sub-arachnoid tissues, and there was a small abscess in the substance of the brain. Pus was found in the diploë, and in the superior longitudinal sinus, and there was puriform effusion in both pleuræ, with purulent infection of both lungs. The other organs were quite healthy.

I have never known the trephine applied at St. George's Hospital with the view of evacuating matter situated either under the dura-mater or in the brain. In one case, in this decennium, matter, it is true, was let out of a large abscess in the brain, but then the operation was not undertaken with this view. It was thought, from the various symptoms, that the matter would be found immediately under the bone.

A man, æt. 25, was admitted into St. George's Hospital, under the care of Mr. Cæsar Hawkins, in November, 1847, with a small scalp-wound, all but healed, and a narrow sinus leading down to an exposed portion of the left parietal. He had met with the injury three weeks before his admission into the hospital, by a blow from a shovel, in a drunken riot. Notwithstanding the accident, this patient had been about

his work as usual, and had only left off on account of great pain in the head, accompanied by a rigor, which had come on the day before he was admitted into the ward. These symptoms were followed by partial paralysis of the right portio-dura, with the tongue drawn somewhat to the left side; and numbness and partial loss of power of the right arm soon supervened. Brisk purgatives were administered, and the untoward symptoms all but disappeared; they soon returned, however, with more intensity, and the right side became completely paralysed. The scalp-wound was at once enlarged, and a trephine was applied over the exposed portion of bone, this was on the eighth day after his admission; some very foul matter with brain-like substance was evacuated, and two other crowns of a trephine were applied, as the dura-mater was found to be in a sloughy state, with an opening of the size of the thumb, through which the matter escaped from the deeper parts. The relief was but temporary; the patient became perfectly insensible, notwithstanding the free discharge of the matter; the dura-mater sloughed more extensively, and the brain protruded through the opening; death took place five days after the operation. The following were the post-mortem appearances. The bones of the skull were healthy; the dura-mater was in a sloughy condition, with a large opening, through which the cerebral substance was protruding, and, in the centre of this protrusion, there was a small opening leading into a large, foul abscess, which occupied the back part of the left hemisphere, and extended down to the lateral ventricle, into which it had all but burst. The abscess was perfectly circumscribed, but the surrounding parts of the brain were soft and pulpy, and of a pale lemon colour; these appearances extended nearly over the whole of the left hemisphere. The septum lucidum was softened, and easily torn, and the lateral ventricles were distended with clear serum. The cavities of both pleuræ contained bloody serum, and there was congestion of both lungs. The other viscera were healthy.

Sometimes, when the trephine is applied over a piece of

exposed and discoloured bone, it happens that matter is observed to proceed from the cut surface of the bone, before the dura-mater has been reached. The quantity of matter, under such circumstances, may be sufficient to flow up, and may lead to the supposition that the skull has been perforated long before it has been so. Lately, I witnessed a well-marked example of this; and, after the removal of the circle of bone, I distinctly traced the matter pouring out of several large holes, which proved to be the openings of the large venous channels contained in the diploë.

Purulent infection was observed in 14 out of the 23 fatal cases of scalp-wound, and, although developed in other injuries, in none is it more frequently so than in those of the head, and that, too, in cases where the injury has apparently been of a trivial nature. The well-known fact that this disease is found especially in injuries involving the osseous system, will serve to explain the frequency of the development of this most formidable complication in accidents about the head, where the bones are not only abundantly supplied with cancellous tissue, but where are also found venous canals much larger and much more numerous than in any other part of the skeleton. That no cases of this kind should have fallen under Pott's notice is surprising, and the more so, as Desault and others were at about the same period directing the attention of surgeons to this affection, as one of the most common consequences of injuries of the head.

It has been thought by some foreign pathologists, that the early application of the trephine made by Pott and other surgeons, in cases of exposed and contused bone, might, in some measure, serve to explain why these practitioners had not met with more cases of purulent infection in injuries of the head. M. Chassaignac especially thinks, that the removal by the trephine of the contused bone before suppuration has taken place in its diploë, destroys the source from whence the secondary mischief is for the most part derived; but such an explanation can scarcely be admitted as a valid one; for in how many cases of purulent infection

after amputation do we not find extensive suppuration in the cancellous tissue of the bone?—cases in which there had been no injury, and in which suppurative inflammation did not exist in the bone previous to the removal of the limb.

Section II.

Fractures and Separation of the Sutures.

In this decennial period, 78 cases of fracture of the skull were examined; 18 of which were complicated, with other severe injuries, which, in most instances, would of themselves have been sufficient to cause death.

Of these 78 cases, 56 were simple fractures, and 22 were compound: of the simple fractures, 19 were, moreover, accompanied by wounds of the scalp, not exposing the bone.

Extensive separation of the sutures coexisted with the fractures in 14 cases.

In 47 cases, the injury had been produced by the patients having fallen from various heights; in 10, the blow on the head had been inflicted by some heavy instrument; in 4, the injury had been caused by a cart-wheel passing over the head; in 3, a sharp instrument had penetrated through the bones of the skull; and in 15, in which the injury to the bones was very extensive, no accurate account of the nature of the accident was given.

In the 56 cases of simple fracture of the skull, there was only one single instance in which the injury was confined to the spot upon which the blow had been struck, the upper part of the skull. In this case a piece of bone, somewhat larger than a sixpence, and comminuted, was driven in below the level of the surrounding parts, which presented no other traces of injury. The patient, a man forty years of age, having been thrown from his coach-box, had struck his head against the ground, and remained in a state of insensibility for some little time; having rallied, he went about his work as usual, and the fracture was not detected until some months afterwards, when symptoms of cerebral irritation made their appearance.

In the 22 cases of compound fracture of the skull, it was found, on the other hand, that the injury to the bones was in 9 cases strictly limited to the original seat of the injury. Of these 9 cases, 1 was a mere fissure; 5 were accompanied by depression; and 3 were caused by a sharp instrument. Two of the latter cases presented a somewhat unusual appearance; a small piece of bone having been partially detached was bent upwards, and thus raised two or three lines above the level of the skull; the fragment was, however, immoveable, as it was still connected at one part to the surrounding bone, the external table of which was at this part only partially fractured: in one instance, the injury had been produced by a chisel falling from a great height on the head; in the other, the patient had fallen from a great height, with his head upon some iron railings, one of the spikes of which had penetrated through the bones.

Fractures of the base of the skull seldom exist alone; in a very large proportion of cases this injury coexists with fractures radiating from the point where the blow was struck. In 68 cases of fracture of the base, 6 only were confined to this region; and even of these, it was only in 2 cases that no traces of fracture could be detected at the seat of the blow. In a man, twenty-three years of age, with two bruised scalp-wounds, denuding the bone at the vertex, the left orbital plate was the only part where a fracture existed; it was a mere fissure, and no blood was extravasated in the neighbourhood. In the second case, a man, thirty-six years of age, whilst carrying a heavy piece of timber, fell backwards, and struck the back part of his head on the pavement, the piece of timber at the same time falling on his forehead. No fracture was detected either in the occipital, or in the perpendicular portion of the frontal, but the body of the sphenoid presented an extensive and comminuted fracture, from which linear fractures were traced in various directions; one fissure ran forwards into the cribriform plate of the æthmoid, and slightly into the left orbital plate of the frontal; another, passing outwards, reached the squamous suture; and a third passing outwards and backwards, along

the anterior surface of the petrous portion of the temporal, was traced as far as the squamous portion. In the remaining 4 cases, which were confined to the base, the patients had fallen on the back of the head, and the line of fracture, starting from the point which had been struck, passed perpendicularly into the foramen magnum.

In civil hospitals, a very large proportion of the fatal fractures of the skull exhibit more or less extensive injury of the base, radiating from the upper or lateral parts. Of the 78 fractures of the skull which died in St. George's Hospital in the space of ten years, 62 cases presented fractures commencing at the upper or lateral parts, and spreading into the base. In such cases the extent of the injury is easily accounted for, by the nature of the accident which usually befalls the patient; a fall from a great height, or a blow from some heavy instrument, is in a large majority of instances the cause of the fracture.

In 51 cases, the fracture was simply linear; and in 27, it was in some part of its course comminuted. This comminuted appearance existed for the most part at the seat of the blow in the compound fractures; but in the simple ones, it was mostly at the base.

In 6 cases of simple fracture, the injury was accompanied by depression, which in all was very slight. In 10 cases of compound fracture there was also depression of the fragments; in 1, this depression was very slight; but in the other 9, it was considerable. In 4 cases of fracture of the base, some fragments of bone had been displaced, and torn through the membranes; this displacement of bone, with laceration of the membranes, is rarely met with in fractures of the base. The following are the 4 cases in which it occurred. In a man, twenty-seven years of age, who had fallen from a scaffold thirty feet high, there was a laceration of the dura-mater, corresponding to a comminuted fracture of the upper wall of the left orbit. A man, thirty years of age, presented a comminuted fracture of the petrous portions of the temporal bones, with a laceration of the dura-mater on the anterior surface of the left petrous portion com-

municating with the tympanic cavity, and there was also a laceration of the dura-mater corresponding to the basilar portion of the sphenoid, through which a probe was readily passed into the sphenoidal sinuses and into the nostrils. In a man, twenty-nine years of age, who had fallen from a height of fourteen feet, several fragments of the æthmoid and sphenoid were driven through the membranes, and into the substance of the brain. A man, age unknown, whose head had been crushed by a cart-wheel, presented a laceration of the dura-mater, corresponding to a fracture of the basilar-bone, through which an escape from the posterior nares had taken place into the cavity of the arachnoid.

Fractures of the skull, with depression of the inner table alone, occur, I think, but very rarely. In the 78 cases of this decennium, it was met with three times, in two of which the depression was very slight, so slight that it might easily have escaped notice. The third instance, however, afforded a well-marked example of this kind of injury; the fracture of the inner table was extensive and comminuted, and the depression of the fragments was considerable, but the outer table presented only a slight trace of injury. I have never met with but one other case of this kind.

Practically, the skull may be divided into three different zones or segments; an anterior zone, formed by the frontal, the upper part of the æthmoid, and the fronto-sphenoid; a middle zone, formed by the parietals, the squamous, and the anterior surface of the petrous portions of the temporals, with the greater portion of the basi-sphenoid; and a posterior zone, which is formed by the occipital, the mastoid, and the posterior surface of the petrous portions of the temporals, with a small part of the body of the sphenoid.

Fractures of the skull, beginning at the seat of the blow, and from thence spreading into the base, are oftentimes, it will be found, limited exactly to the bones belonging to each of these three different zones. In 25 cases, where the injury was thus strictly limited, the line of fracture was in 5 cases traced from the forehead into the bones at the base of the anterior zone, and into these alone; from the parietals,

or temporals, in 14 cases, the fracture was found to pass into the corresponding bones at the base; and in 6 cases, the line of injury passed from the occipital into the posterior segment only. In the cases belonging to the anterior zone, the fracture passed perpendicularly in 3; in 1, it was oblique from side to side, and in 1, it was extensively comminuted. In the middle zone, the fracture was confined to one side in 12 cases, and in 2, it occupied both sides; in 1 of these there had been two distinct accidents, at an interval of a week, and there were two separate fractures, each limited to one side; and in the other case, the fracture was accompanied by separation of the coronal suture. In the posterior zone, the injury in 5 cases passed longitudinally, and in 1 there were several lines running in different directions. The results of this analysis certainly corroborate some of the views so ably advocated by Dr. Aran.[1]

Frequently, however, no such exact limits can be assigned to the extent of the fracture, and two of the fossæ at the base may be involved at the same time: wedged in between the other two fossæ, the middle one, in such cases, either receives or transmits the injury. In these cases, the fracture generally runs obliquely from one fossa into the other, or else, coursing along the boundaries of the regions in the neighbourhood of the sutures, which may be widely separated, it passes into the fossæ. In 29 cases, where the line of fracture occupied two fossæ at the same time, it was found in 14 cases in the middle and anterior fossæ, and in 15 cases in the middle and posterior ones. Separation of the sutures not unfrequently accompanies this form of accident. In the cases belonging to the middle and anterior fossæ, the fracture was transverse, and occupied both sides in 7, with extensive separation of the coronal suture in 3; in 6 cases, the fracture passed more or less obliquely from side to side, and in 1, it was limited to one side. In those cases belonging to the middle and posterior fossæ, the fracture was transverse, and occupied both sides in 6 cases, in 5 of which it was accompanied by separation of the lambdoid suture, and in 1 by separation

[1] Archives Générales de Médecine, 4e série, t. vi.

of the sagittal suture; in 4 cases the injury passed obliquely from one side to the other, and in 5, it was confined to one side.

Much less frequently does it happen that all three fossæ at the base are fractured together, and, as might be supposed, it generally only happens in those cases where the patients have either fallen from a very great height, or where the blow has been of a very severe character. All three fossæ were implicated at the same time in 10 cases, in 6 of which the fracture was transverse, and occupied both sides, with separation of the sutures in 4; and in 4 cases, it was limited to one side.

Fractures involving the middle zone, either by itself, or in combination with the others, are by far the most common of all fractures of the base. The middle fossa presented more or less extensive injury in 53 cases.

Transverse splitting of the skull is much more common than the longitudinal one. In 2 cases only was the skull thus split longitudinally, from the forehead to the foramen magnum. In both, the upper part of the forehead had first struck the ground, the patient having fallen several feet, and in both, there was also separation of the sagittal suture.

Modern researches have not only proved that injuries of the skull implicating the base may frequently be readily recognised, but they have also proved that the exact course of the line of fracture may not unfrequently be clearly diagnosed.

Fractures of the base of the skull, involving the orbital plates of the frontal bone, are oftentimes accompanied by an effusion of blood into the orbit and eyelids, which may become a sign of great value in the diagnosis of fractures of this region. In this decennium, the upper wall of the orbit was more or less extensively broken in 23 cases. In 8 of these cases there were no external marks of injury about the orbital region; in 5 cases, the effusion of blood occupied the eyelids only; but in 10 cases this effusion of blood occupying the eyelids and the cellular tissue of the orbit, gave a clear diagnosis of the nature of the injury.

In making use, however, of the bruised appearance of the eyelids as a means of diagnosis of fractures of the base, not only should the skull be carefully examined as to the spot which was struck, but especial attention should be paid to the state of the sub-conjunctival cellular tissue of the eye-ball, which will always be ecchymosed, if the blood comes from the bottom of the orbit. In such cases, the blood proceeding from the back part of the orbit makes its way forward, and at first presents itself under the ocular conjunctiva. If the patient be seen at a very early period after the accident, or if the effused blood be in small quantity, it may be altogether confined to the conjunctiva, where it first becomes visible, but this I have only seen in a very few instances; generally the blood has been effused in the eyelids, as well as upon the eye-ball. I cannot, however, quite agree with M. Velpeau in thinking that the lower lid always becomes discoloured before the upper one; that the blood does most frequently gravitate into the lower lid, there is no doubt; but I have several times seen the upper lid and sub-conjunctival cellular tissue ecchymosed, without any discoloration of the lower one. It sometimes happens, that the effusion of blood into the orbit is so extensive, that the eye-ball becomes protruded; in such cases, the fracture has been most extensive. Protrusion of the eye-ball occurred in 3 cases out of the 10 in which blood had been effused into the orbit.

Bleeding from the ear, in severe injuries of the head, is of not unfrequent occurrence, and becomes one of the most valuable diagnostic signs of fracture of the base, running through the middle fossa, and implicating the petrous portion of the temporal bone. Should this bleeding be extensive, and continue for some time, and should it be ascertained that there is a rupture of the membrana tympani, there can be no doubt, whatever may be the issue of the case, that the cavity of the tympanum has been laid open, and a communication established between it and some of the numerous vascular channels which surround the petrous bone, or with an extravasation of blood within the cranium. In 32 cases

of fracture of the middle fossa implicating the petrous bone, the tympanum was thus laid open, and its membrane ruptured in 15, or very nearly one half; the flow of blood in most of these cases was profuse and continuous, and, in all, the diagnosis of the injury was clear. Fractures of the temporal bone, however, may and do frequently occur, in which there is no sign that can lead to the supposition of such an injury. In such cases, either the line of fracture does not extend into the tympanum, or, if it does, the membrane is not ruptured, and the blood cannot consequently get into the external meatus. Fractures of the temporal bone, not involving the tympanum, existed in 12 cases; and in 5 cases, in which the tympanum was fractured, the membrana tympani was not ruptured.

But, of the signs indicative of fracture of the petrous bone, one of the surest is the copious discharge of a watery fluid from the ear, which may or may not be preceded by bleeding. In the majority of cases of this kind which I have seen, a large flow of blood preceded that of the watery fluid. In this decennium, there were only three instances in which there was a discharge of a watery fluid from the ear, after a severe injury of the head.

A man, æt. 42, having fallen off a ladder whilst lighting a lamp, was admitted into St. George's Hospital in August, 1844, under the care of Mr. Cutler, with violent bleeding from the left ear, and other symptoms of extensive fracture of the base. The flow of blood became less and less, and was ultimately succeeded, on the third day, by the discharge of a watery fluid, which continued two days, at the end of which the patient died, having had paralysis of the facial nerve shortly before his death. Among the various lines of fracture at the base, a fissure was traced from the left parietal through the squamous portion of the temporal into the petrous portion, which it cut across at its middle; and, passing backwards into the foramen lacerum posterius, reached the foramen magnum. The tympanum was laid open, and the membrane extensively ruptured. Blood had

been extensively effused between the bone and the dura-mater, principally in the region of the anterior inferior angle of the parietal.

A man, æt. 46, having fallen from a cab, was admitted into St. George's Hospital, under the care of Mr. Cæsar Hawkins, in August, 1849, with extensive hæmorrhage from the left ear, and slight bleeding from the nose. The bleeding from the ear gradually subsided, and was succeeded on the second day by the discharge of a slightly coloured, watery fluid, which became less and less tinged, and ultimately quite clear: it lasted two days, and the patient lived eight days after the accident, having latterly had slight paralysis of the left facial nerve. On the left side, a fissure was traced from the back part of the parietal through the mastoid portion of the temporal, and along its petrous portion into the spheno-temporal suture, traversing the tympanic cavity, and rupturing its membrane. There was little or no blood in the line of the fracture, but the cavity of the tympanum was filled with lymph.

A man, æt. 23, who, it was said, had received a blow on the right side of the head, was admitted into St. George's Hospital, under the care of Mr. Cutler, in September, 1850, with violent bleeding from the right ear, which, on the following morning, was succeeded by a watery fluid, blood-tinged: he died sixty hours after the accident, the escape of fluid from the ear continuing up to the time of his death. With an extensive fracture passing from the right parietal, in front of the zygoma, into the middle fossa, and thence across into the left anterior fossa, a small fissure was also traced out extending obliquely along the anterior surface of the petrous bone into the foramen ovale and foramen rotundum: the cavity of the tympanum, which was filled with blood, was laid open, and its membrane ruptured at its anterior part. About half an ounce of blood was found between the bone and the dura-mater, in the temporal region.

The copious discharge of a watery fluid from the ear in fractures of the base of the skull has, of late years, greatly

occupied the attention of surgeons, and many and ingenious are the explanations which have been given as to the source of this fluid; but, of all these explanations, there is none, I think, so satisfactory as that which assigns the source of this discharge to the escape of the cerebro-spinal fluid through a fissure of the meatus internus, and a laceration of the corresponding part of the dura mater, by means of which a communication is established between the cavity of the tympanum and the sub-arachnoidean cellular tissue in contact with the seventh pair of nerves; this must also be accompanied by a rupture of the membrana tympani.

That such is the origin of this watery discharge in many instances has been most clearly demonstrated by M. Robert[1] in his valuable paper, in which it appears to me, however, that the author has laid rather too much stress upon the direction of the fracture observed in his cases.

All that is wanted for the cerebro-spinal fluid to escape from the ear is, that the loose sub-arachnoidean cellular tissue which accompanies the tubular sheath of the arachnoid, belonging to the seventh pair of nerves, into the meatus internus, should be made to communicate with the cavity of the tympanum, and with the external ear through a rupture of the membrana tympani. Such a communication may be brought about, not only as stated by M. Robert, by a transverse fracture cutting the petrous bone across its middle, but also, I believe, from my own observations, by a fracture running along the anterior surface of this bone, in a more or less zig-zag direction, from the apex to the base. A fracture following either of these two directions may catch the meatus internus in some part of its course, and also run into the cavity of the tympanum; in addition to a fracture following such a course, the dura-mater lining the meatus must be ruptured, but the arachnoid accompanying the nerves may or may not be lacerated.

In the first of the three cases mentioned above, the fracture was a transverse one, and cut the petrous bone across its middle; but, in the other two cases, the line of fracture

[1] Mémoires de la Société de Chirurgie de Paris, vol. i.

ran along the anterior surface of the bone, and was more or less longitudinal in its direction.

In the following two cases which have lately fallen under my notice, the relation of the fracture to the meatus internus was traced out; and in the latter case a careful dissection, which I subsequently made, clearly revealed the extent of the injury and its exact nature.

A man, æt. 65, having been knocked down by a mail-cart, was admitted into St. George's Hospital, under the care of Mr. Keate, with severe contusion of the scalp on the left side of the forehead, and a small wound, exposing the bone, in the neighbourhood of the left superciliary ridge, but no fracture could be detected at this spot. The eyelids were closed by the effused blood, and there was profuse hæmorrhage from the right ear, and from the nose: the collapse was extreme, with perfect insensibility, and the mouth was drawn to the left side. On the following day the bleeding from the ear continued, and the sub-conjunctival cellular tissue of the lids and orbit was extensively infiltrated with blood. The bleeding from the ear gradually diminished, and was followed by a watery fluid, blood-tinged, which subsequently became quite clear. The patient lived eleven days in a semi-comatose state, and the discharge of watery fluid continued up to the time of his death. A line of fracture was traced from the left frontal, through the orbital plate, across the æthmoid and body of the sphenoid, into the right petrous bone, along the anterior surface of which it ran from the apex to the base, passing in the line of the bottom of the meatus internus; the cavity of the tympanum was laid open, and the membrana tympani was ruptured. In this case, there was no transverse fracture cutting across the petrous bone.

A man, æt. 52, was admitted into St. George's Hospital, under the care of Mr. Cæsar Hawkins, in March, 1852, with a profuse discharge of a clear watery fluid from the left ear. The patient having, it appeared, fallen four days

before his admission, in some kind of a fit, had struck the back part of his head against a stone pavement; he had been insensible for an hour after the accident, and, after recovering, had immediately noticed the discharge from the ear, which had continued ever since, and had not been preceded by any bleeding. There was a small scalp-wound, not exposing the bone, on the right side of the occipital, near the lambdoid suture, but no fracture could be detected in this or any other part of the skull. The watery fluid, of a very faint pinkish hue, dropped freely from the ear, the flow being increased when the patient was speaking; there was great pain in the head, and the patient's manner was oppressed and slow; the countenance was heavy, and of a dusky colour; the hearing, on the left side was much impaired, in fact all but lost, but there was no paralysis of the facial nerve. The patient was perfectly sensible, with a pulse laboured, and at 56. Symptoms of diffuse inflammation of the membranes soon made their appearance, and the man lived only four days after his admission into the hospital, the discharge of watery fluid having continued, with but little variation, up to the time of his death. A line of fracture was traced from the neighbourhood of the right lambdoid suture, obliquely through the occipital, to the left side of the foramen magnum: proceeding from thence through the lateral sinus, close to the foramen lacerum posterius, it reached the petrous portion of the temporal, which it cut across between the meatus internus, and the superior semi-circular canal, and having passed about midway through the thickness of the petrous bone, the fracture made a sudden bend, and then proceeded along the anterior surface of this bone towards the inner opening of the carotid canal, where it terminated. In this course the fracture at its bend just caught the bottom of the meatus internus, and, passing through the vestibule, reached the tympanum, the cavity of which was widely laid open. In addition to this fracture there was a fissure passing perpendicularly through the cribriform plate at the bottom of the meatus, and here, too, the dura-mater lining this channel

presented a corresponding laceration. The membrana tympani was ruptured in two different places; one of these ruptures was close to its outer margin, and the other, and larger of the two, was in the centre. There was no laceration of the lateral sinus, but the cavity of this channel, from its mastoid portion to the jugular vein, was blocked up by a firm coagulum of a rusty brown-colour, and adherent to the inner coat. No laceration was detected in any part of the dura-mater, save that which was at the bottom of the meatus internus. Extensive marks of inflammation, of a diffuse character, were found about the membranes of the brain.

In this case, previous to the dissection of the fracture, a slender pig's bristle, having been gently passed to the bottom of the meatus, and immediately above the nerves, made its way into the tympanic cavity, from whence, with a little management, it was brought through the rupture in the membrana tympani, thus plainly showing the course the cerebro-spinal fluid had taken.

It has been stated by many surgeons abroad that most of the cases of this watery discharge in fractures of the base occur in adults, or in very young persons; but, in the cases above described, the ages were 42, 46, 23, 65, and 52, and nearly all the cases of this kind, which have fallen under my own notice, have been in persons beyond the adult period of life. The meatus internus being larger, comparatively speaking, in the child than in the adult, may have favoured this idea; but a careful examination of a great number of skulls, fully developed, will prove that the size of the meatus varies very much in different heads; in some being very small, a mere chink, and very shallow, whereas in others it is of a large size, and deep. Such variations must give rise to a greater or less facility for the escape of the cerebro-spinal fluid, should a fracture occur in this part.

A close examination of these cases of watery discharge will show that this fluid makes its appearance under different circumstances. In some cases, no discharge of blood pre-

cedes that of the watery fluid, which from the beginning is all but limpid; in others, a copious bleeding precedes, and, even in these latter, the watery fluid may either imperceptibly follow the bleeding, or a certain interval, two or three days, may intervene between the bleeding and the watery discharge. Such differences may perhaps be thus explained; in the first instance, it is evident that no vascular channel has been laid open; in the second, the flow of blood masks for a time the watery fluid, which becomes more and more apparent as the blood ceases to run; in the third, the fissure being a very slight one, the clotting and drying of the blood in the tympanum and in the internal ear stop the course of the watery fluid, which gradually makes its appearance as the clots become detached. In four out of the five cases mentioned above, copious bleeding preceded the watery fluid.

M. Chassaignac[1] has endeavoured to prove that this fluid owes its origin to the filtering of the colourless part of the blood, circulating in the lateral sinus, through a minute laceration of the outer wall of this channel, caused by a fracture occupying this part of the temporal, and running through the petrous bone into the cavity of the tympanum. In addition to several other objections which may be urged against M. Chassaignac's explanation, it is impossible that, in the last-mentioned case, any fluid could have filtered from the blood circulating in the lateral sinus, as this channel was completely blocked up by coagula, evidently of some days' standing.

It has been thought by some surgeons that the watery fluid proceeds from the cavity of the arachnoid, but such cases, if they do occur, must be very rare. In this decennial period, there was only one case in which the requisites existed for this to have taken place, even had the patient survived long enough. With a comminuted fracture of the anterior surface of the petrous bone, immediately corresponding to the upper wall of the tympanum, were two small openings through the dura-mater and its arachnoid, produced by some pointed fragments of bone; the tympanum

[1] Mémoires de la Société de Chirurgie de Paris, vol. i.

was widely laid open, and its membrane torn across. In such a case, it is possible that the arachnoid, irritated by the spicula of bone, might pour out a quantity of fluid which would find its way into the external meatus.

In these remarks on the source of the watery discharge from the ear after an injury to the head, I wish it not to be inferred that I think this discharge is, in all cases, due to the escape of the cerebro-spinal fluid. That it is so in most cases, and especially in those where the fluid is abundant, and from the first all but colourless, further researches, such as those lately published by Mr. Hilton,[1] will, I have no doubt, fully demonstrate. Careful observations and dissections may, however, prove that this is not the only source of this watery discharge; but in all future researches into this subject, the exact line of the fracture, and its precise relation to the meatus internus in the whole of its length, must be most clearly made out.

In some few instances, the watery fluid is succeeded by a thin, sero-purulent secretion, which becomes thicker, and ultimately puriform: when this takes place, it is that suppuration has been established along the line of the fracture of the petrous bone. Puriform lymph was in one instance found along the line of fracture, and in the cavity of the tympanum in this decennium.

Bleeding from the nose or mouth, or vomiting of blood, not unfrequently occurs after injuries of the head, but the great vascularity of the membranes lining these cavities renders this bleeding much less valuable as a diagnostic sign of fracture of the base, than that which proceeds from the ear. Still, if the bleeding be copious, and continue for some time, there is no doubt that this symptom may also become one of great value. In 32 cases of fracture of the base, implicating the central bones of this region, bleeding from the nose or mouth, oftentimes most profuse, or subsequent vomiting of blood, occurred in 14 instances, in 4 of which the fracture was confined to the æthmoid; in 3, it was confined to the body of the sphenoid, and in 1, to the basilar

[1] Clinical Lectures delivered at Guy's Hospital. (Lancet, 1853.)

process; in 5 cases, the æthmoid and sphenoid were involved at the same time, and in 1 case all these three bones were fractured.

Cases of united fracture of the base are but seldom met with; in this decennial period there was one case in which union, partly osseous and partly fibrous, had taken place in an extensive fracture of this region. It occurred in a man, æt. 53, who was admitted into St. George's Hospital, under the care of Mr. Cutler, in 1849, with a severe injury to the head, which he survived two months, and then died of erysipelas of the scalp. A line of fracture, commencing in the neighbourhood of the right parietal eminence, passed behind the external angular process, and, extending through a small portion of the great wing of the sphenoid, reached the back part of the orbital plate of the frontal, from whence it was traced through the lesser wing and body of the sphenoid, in front of the olivary process, and terminated at the inner part of the left optic foramen. This extensive line of fracture was for the greater part filled up by a deposit of new bone, and, in the situation of the lesser wing, so complete was the bony union, that all trace of the fracture had almost disappeared; but, in other parts, and especially across the body of the sphenoid, the only bond of union was a dense layer of fibrous tissue. An irregular deposit of new bone, spongy and vascular, was found on the inner side of the frontal, spreading some distance from the line of fracture, which it in some places completely overlapped.

Extensive separation of the sutures coexisted with the fractures in 14 cases, in several of which two or more sutures were implicated at the same time. Separation of the coronal suture occurred in 7 cases; in the lambdoid suture, this separation occurred in 6 cases; in the sagittal suture, there were 4 cases; in the petro-occipital, there was 1; in the temporo-parietal, there was 1; and in the spheno-parietal, there was also 1. In one case, where there was complete separation of the coronal suture, the frontal was, at the same time, extensively separated from its connections with the other bones of the cranium.

In one case, and in one only, was there a separation of a suture without a fracture; it occurred in the posterior part of the squamo-parietal suture. The temporal having been slightly separated from the parietal and driven upwards, presented, at first sight, the appearance of a fracture with depression of bone.

I cannot conclude this part of my communication without offering my warmest thanks to my colleagues, the Medical Officers, the Curators of the Museum, and the Registrars of St. George's Hospital, whose Cases and Notes have been, in the most liberal manner, placed at my disposal.

A CASE

OF

PERFORATING ULCER OF THE ŒSOPHAGUS,

WHICH

CAUSED DEATH BY PENETRATING THE AORTA.

BY

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COMMUNICATED BY

CAMPBELL DE MORGAN, Esq.

Received April 15th.—Read June 14th 1853

W. S—, a painter, æt. 51, was admitted into the Middlesex Hospital, under the care of Dr. Hawkins, at 6 p.m., November 19th, 1852. He was a tall, well-made, muscular man; his countenance death-like; pulse scarcely perceptible; extremities cold, and was not conscious when spoken to. It was stated by those who brought him, that he had left his work an hour previously, and was shortly afterwards attacked with profuse hæmorrhage from the mouth, the blood being bright, red, and frothy, immediately after which he fell senseless, and remained in that condition until seen as above described.

On inquiry into his previous history, his son declared that he had been always of very temperate habits; but for some time past had been desponding, in consequence of having but little work; that a week ago he first complained of a deep-seated pain at the top of the sternum, shooting through to the spine, for which he applied for medical advice, and was said to be somewhat relieved.

The patient was immediately placed in bed; ice was put

into his mouth, and large doses of acetate of lead administered at frequent intervals, and attempts to bring on reaction used with some success, for on the following morning at 9.30, the pulse was 100, firm and steady, though small. On auscultation, free respiration was heard in every part of the chest, except the upper lobe of the left lung, where the respiratory murmur was deficient, the vocal resonance increased, and there was also some dulness on percussion.

He now stated that he had had a slight cough for many weeks, but without expectoration, and that previous to this attack he was of his usual vigour, and had not lost flesh. At 10 a.m., whilst in the recumbent posture, there suddenly gushed from his mouth upwards of a pint of bright arterial blood, with which was a loose coagulum of the size of a small lemon; while this occurred, the respiration continued unimpeded. Some wine was poured into his mouth, but he was unable to swallow, and a few minutes afterwards he died.

There had been no action of the bowels from the time of his admission, but during the fatal vomiting the bladder involuntarily emptied itself.

My acquaintance with the case having commenced at the post-mortem examination, I am indebted for the above particulars to Mr. Corfe, the Resident Medical Officer at the Hospital.

The body was examined on the 22d of November, fifty-three hours after death; it was found to be well made and muscular, not at all emaciated; the skin was blanched, and the cadaveric rigidity well marked.

On opening the cavity of the thorax, some old adhesions were found at the apices of both lungs; several small crude tubercles in the upper lobe of the left, and the same condition in the right, but to a less degree; the remainder of the lungs were generally normal in appearance, of a pale blueish colour, exsanguine, the anterior margin of both slightly emphysematous; the lining membrane of the trachea and bronchial tubes pale, containing a little mucous froth, not stained with blood.

The heart was full-sized and loaded with fat; the thick-

ness of the walls and dimensions of cavities normal; the muscular structure apparently healthy; the mitral and tricuspid valves slightly thickened, the semi-lunar valves healthy; no coagula in any of its cavities.

The stomach was large and distended, and when opened was found to contain a clot of blood, which formed an exact cast of the interior of the organ; the mucous membrane healthy in structure, but stained with blood; round the cardiac orifice were four irregular-shaped patches of erosion, or rather fissures in the mucous membrane, involving partly the stomach, and partly the œsophagus, their longitudinal diameter in the direction of the tube, and varying from 2 to 3 lines in length.

In the mucous membrane of the œsophagus, 3 inches above the cardiac orifice, on the left side, was a perfectly circular opening, $3\frac{1}{2}$ lines in diameter, with slightly elevated edges, which were sharp and clean, as if cut out with a punch, and beveled off from within outwards, the mucous membrane being destroyed, to a greater degree than the tissues beneath, in fact, exactly resembling, on a small scale, the circular perforating ulcer so frequently met with in the stomach: this was immediately surrounded by a reddish halo, but the mucous membrane of the œsophagus elsewhere appeared healthy; on the inner surface of the aorta, at the termination of the descending portion of the arch, on the right side, nearly corresponding with the ulcer in the œsophagus; but on rather a higher level, was an irregular opening, about $1\frac{1}{2}$ lines in width, with ragged edges formed by the projecting and torn lining membrane; extending forwards from this, for a distance of 3 lines, the coats of the aorta were considerably thinner than elsewhere, the lining membrane over this thin portion being, however, continuous and healthy, and transmitting the dark colour of the stained tissues beneath; about half an inch above, was a patch of opaque atheromatous deposit, the centre of which had advanced to calcification; there were also several other small patches of white deposit in other parts of the lining membrane of the aorta. The opening in the aorta and that

in the œsophagus communicated freely, a probe passing readily from one to the other; the distance between the inner surface of each tube was 7 lines, the passage between them contained a clot of blood, and was surrounded by loose cellular tissue infiltrated with blood, but presenting no signs of inflammation, or any attempt at adhesion between the vessel and the œsophagus.

The duodenum contained a loose coagulum, resembling that in the stomach, the jejunum was pale, empty, and contracted; its lining membrane pale and unstained. The ileum and whole of the large intestines down to the sphincter ani were distended with semi-fluid blood of a perfectly black colour, with scarcely any fæcal matter; the mucous membrane stained, but presenting no trace of inflammation or ulceration; the Peyer's patches and other glands normal. The mesentery and omentum healthy and well loaded with fat; the liver, spleen, and kidneys of natural size, pale, but apparently healthy in structure; all the other organs and tissues healthy and normal.

Cases of communication between the aorta and œsophagus are to be met with in various works on pathological anatomy; most of these have their origin in aneurism of the vessel; others are said to arise from ulcerations of the œsophagus, generally of either a tuberculous or malignant character, or as in one related to me by Mr. Henry, caused by a stricture of the latter organ; but I believe that detailed cases exactly corresponding to the one above described are rare, if not altogether wanting.

From a careful examination of the specimen, and particularly of the character of the two openings, I cannot but conclude that the ulcerative process commenced in the mucous membrane of the œsophagus, and spread through the intervening cellular tissue to the aorta, the outer coats of which appear to have been considerably destroyed before the lining membrane gave way. The existence of atheromatous deposits on the interior of the latter vessel, might incline to the supposition that it was here that the lesion originated; but this condition is so frequent in persons

above the age of 50, that it may have been only a coincidence, especially as the lining membrane immediately surrounding the opening appears healthy, and there is no appearance of separation of the coats of the artery or dissecting aneurism, which usually occurs after spontaneous laceration of the internal coat. Moreover, the former conclusion is strongly supported by the regular form and sharp and bevelled appearance of the edges of the ulcer in the œsophagus, which are not such as are found in ulcers caused by external influence, but, as before stated, exactly coincide, as does the symptom of constant pain in the part, with those ulcers of apparently spontaneous origin, termed by Cruveilhier the "simple chronic ulcer of the stomach," and by Rokitansky the "perforating gastric ulcer," and respecting which the latter author states "the pyloric half of the stomach is the seat of the ulcer, it occurs in extremely rare cases only at the fundus; this affection may also appear beyond the stomach in the upper transverse portion of the duodenum, *but it does not occur in the remaining portion of the intestinal canal.*"[1] Under the head of lesions of the œsophagus he does not mention the occurrence of any ulcer similar to that above described. Cruveilhier and Carswell give no more information on this subject. Förster, in his recently published 'Manual of Pathological Anatomy,' p. 165, after mentioning various well-known causes of ulceration of the œsophagus, says, "moreover, according to Albers, an ulcer occurs in the œsophagus analogous to the perforating ulcer of the stomach (the "simple ulcer of the œsophagus," Albers,) which is situated mostly on the anterior wall, against the bifurcation of the trachea, gradually perforates the coats of the œsophagus, and forms communications with the trachea, pleuræ, mediastina, and aorta, and also causes ulcerations of the lungs." As it is not stated in which of Albers' works this is to be found, I presume that his 'Atlas of Pathological Anatomy' is referred to, in the letterpress

[1] Rokitansky's Pathological Anatomy, 'Sydenham Society's Translation, vol. ii, p. 30.

to the second part of which, at p. 204, is a detailed description of the "simple ulcer of the œsophagus," which he says follows an exactly similar course to the simple ulcer of the stomach, and must be distinguished from those caused by external pressure, as from tumour, abscess, or aneurism, and from those spreading ulcers which are the consequence of "*œsophagostenosis*." He states further, at p. 219, "the spreading of ulcers of the œsophagus into the aorta has been several times observed: Van Doeveren relates the case of an old woman in whose corpse a large ulcer of the œsophagus was found, which had destroyed the wall of the aorta; the rush of blood through the ulcer had been so copious that the stomach was found quite distended with it. Sandifort relates precisely the same observation." Unfortunately, Albers does not connect this statement specially with the simple perforating ulcer which he had previously described; but his cases may have belonged to any other variety, as indeed in the one described and figured by Sandifort, which appears to be the same as that of Van Doeveren; there is a very large and irregular-shaped ulcer, apparently scrofulous, and the tissues around appear to be considerably involved in the disease.

Another important point illustrating the essentially different nature of Albers' cases and the one I have brought before the notice of the Society, is that in the former "a most intimate growing together of the œsophagus with the aorta" is described, whereas in the latter there was no trace of any such adhesion.

In conclusion, I trust that the Society may not think that I have trespassed upon its valuable time in calling attention to a case which, from its unique character, appears to be of great pathological interest, presenting as it does an example of an ulcer of the œsophagus of a kind very rarely met with in that situation, and remarkable for having, without spreading in width, advanced rapidly and deeply through a considerable space of loose cellular tissue, and finally terminated its course by perforating a tissue so little liable to ulceration as the coats of a large artery.

ON

SMALL POX AND VACCINATION.

ANALYTICAL EXAMINATION OF ALL THE CASES ADMITTED DURING SIXTEEN YEARS,

AT THE

SMALL POX AND VACCINATION HOSPITAL, LONDON;

WITH A VIEW TO ILLUSTRATE THE PATHOLOGY OF SMALL POX, AND THE PROTECTIVE INFLUENCE OF VACCINATION, IN DEGREES VARYING ACCORDING AS THE VACCINATION HAS BEEN PERFECTLY OR IMPERFECTLY PERFORMED.

BY

J. F. MARSON,

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COMMUNICATED BY

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WITHIN the last few years Smallpox has been several times epidemic in London. Many vaccinated as well as unvaccinated persons have been attacked by it, and the public have become, in consequence, somewhat anxious about their security. I have, therefore, thought that the profession would be interested, and that they might possibly elicit useful information from the leading particulars, arranged and classified, of the patients admitted for sixteen years, at the Smallpox and Vaccination Hospital, London. During the period alluded to—1836 to 1851, inclusive—smallpox has been epidemic four times, viz., in 1838, 1844, 1848, and 1851, besides a short epidemic in the winter of 1840-1. Rather more than half of the patients admitted with smallpox into the hospital, have had the disease after having been vaccinated. The particulars of each patient

were entered at the time in the register of the hospital, in the majority of instances by myself. Having analysed these records with minute accuracy and strict fidelity, I beg now to place the results before the Society.

The analysis will be found to have reference principally to the following points:

1. Natural smallpox.
2. Smallpox after smallpox.
   - *a*. After natural smallpox.
   - *b*. „ inoculation.
3. Smallpox after vaccination.
   - *a*. Number of cicatrices.
   - *b*. Character of cicatrices.
   - *c*. Vaccinated, but without cicatrices.
4. Febrile eruptive diseases mistaken for smallpox.

No one could be long in attendance at the Smallpox Hospital, and fail to be struck by the remarkable difference presented to his notice between the vaccinated and unvaccinated patients, and also between the vaccinated cases themselves, some patients having smallpox in a mild form, wholly devoid of danger, whilst others have it in great severity, scarcely if at all lessened by the previous vaccination. Under these circumstances it became desirable to ascertain carefully, so far as possible, all the antecedents regarding the vaccination of each individual admitted, with a view of discovering, if possible, the cause of this difference, to be able to account for the extreme mildness of the disease in some cases, and the danger and unmitigated course, sometimes death, in others.

Smallpox in the unprotected, remains to this day as virulent as it perhaps ever was, destroying about one third of all whom it attacks, and is especially destructive to infantile life. Vaccination, when performed in infancy, affords almost complete protection against the fatality of smallpox up to the period of puberty. This disease does not usually occur after vaccination until several years have elapsed; at least, such is the general experience of the Smallpox Hospital,

although there have been some instances to the contrary. The particulars, unfortunately, of the progress of the vaccination, as observed by medical men, are but rarely noted at the time, and preserved,[1] so that when variola occurs it becomes necessary to trust to the accounts given of the vaccination by the patients themselves or their friends, and to observe carefully the appearance of the cicatrices left by the vaccination; smallpox occurring in the great majority of instances in persons between 15 and 30 years of age, the vaccination having been, with but few exceptions, performed in infancy. After this lapse of time from vaccination, the most trustworthy evidence we can generally obtain of its perfection is from the cicatrices, and this evidence I shall be able to show is a very good guide to the general amount of protection conferred by vaccination, if not to be depended on even in each individual case, it is so when the observation is extended and applied to the community at large. My intention in bringing this subject before the Society is not to dwell on individual cases, except so far as they form one comprehensive whole; but to look at the subject in its widest grasp, to treat of the cases in the aggregate, by hundreds and by thousands, as they have occurred in hospital practice extended over a series of years. A few exceptional cases could perhaps be found to the rule of practice it will seem desirable to draw from the accompanying records; but these very exceptional cases have been the cause of complicating the subject so much when viewed from a limited range, that the profession have been in some doubt what to do, and what to recommend: to endeavour to set these doubts at rest, and to contribute to the public good by indicating how the practice of vaccination may be improved, is the aim of this communication.

It will, perhaps, be objected, and very properly, that the experience of an hospital, as regards the mortality, is not the best criterion by which to judge of the true value of vaccination. In this I should entirely agree, if the evidence rested alone on the mortality; such, however, is not the

[1] There are records, posted alphabetically, of all the persons vaccinated at the Smallpox Hospital, from 1799 to 1838.

case. No one is more alive than myself to the great amount of evil, as well as the great amount of good attendant on hospitals; of evil from collecting the sick together in large numbers, and I would have been the last to have brought forward the experience gained in an hospital of the protective influence of vaccination, as observed in England, if a tithe of the same amount of information on this particular subject, and collected in the same systematic way, were available from other sources; but, unfortunately, it is not.

Although the inferences drawn on any disease, from the mortality of hospitals exclusively, might fairly be objected to, for the reasons well explained by Mr. Farr, in the 'Third Annual Report of the Registrar General,' 8vo, p. 97, still these inferences are not without their value, as the class of cases reported on can generally be obtained in much larger numbers at hospitals than from any other source, and when allowances have been made, as I have made them, in reporting on the patients, and drawing the averages from them collectively at the Smallpox Hospital, we can arrive very nearly at the truth, the great object I have had before me in undertaking these inquiries. To obtain this point, all the patients have been entirely excluded, in calculating the averages, who were affected by superadded disease, just as much as if they had never been admitted into the hospital at all, and the deductions have been made from those who were seemingly affected by smallpox only. All practical men will readily admit that it is impossible, even in rural practice, to meet with a large number of cases of any one disease unmixed with and uninfluenced by other diseases. The same thing occurs in hospital practice. Patients suffering from other diseases are attacked by smallpox and die, who would recover from smallpox but for the injury done to the constitution by the previous disease; and although a large majority of the fatal cases from superadded disease, at the Smallpox Hospital, has undoubtedly arisen from erysipelas, gangrene, &c.,[1] the same thing, unfortunately, is taking

[1] Observations on Ochlesis, or the Disorder generated by the accumulation of the Sick, by George Gregory, M.D., 'Medical Times,' vol. xix, No. 496, p. 431.

place often at our general hospitals, as well as at the Smallpox Hospital, and has given occasion, within the last few years, for the production of papers on hospital diseases;[1] but, in general hospitals, the evil seems to be of less consequence,—or at all events is less thought of,—than at the Smallpox Hospital, because the deaths, and other results, do not there, as they do at the Smallpox Hospital, interfere with a great scientific and pathological question, namely, the protective influence of vaccination. Assuming, however, the Society will give me credit for being fully alive to these evils of hospitals, and for having used my utmost care to exclude, so far as possible, from my calculations of averages of mortality, all cases affected specially by hospital mischief, as well as other forms of superadded disease, such as phthisis; the several *idiopathic* inflammations of vital organs, as pneumonia; puerperal fever,—a disease closely allied probably to erysipelas, if not identical with it, &c., &c.

A most unfortunate and indirectly fatal complication is gonorrhœa, both in the male and female; to the latter more especially. From the inability of the patients to use ablution, carefully, themselves, to the parts affected, once or twice daily, and from the extremely disagreeable nature of this duty for others to perform for them, the parts are not always kept properly clean, the consequence is they are very apt to become gangrenous; absorption of the gangrenous matter ensues, which is followed by pyæmia, phlebitis, the formation of large abscesses all over the body, in short, the usual train of evils produced by the absorption of a morbid animal poison: these cases have been included, of course, in the returns under the head of gangrene, but they would not have arisen had it not been for the gonorrhœa. Allowances having been thus made, the patients are described, as far as it is perhaps possible to describe them, as suffering from

[1] Cases of Hospital Gangrene, and of diffuse Inflammation of the Cellular Membrane, which occurred in St. Bartholomew's Hospital, in December, 1846, and January, 1847, by Holmes Coote, Esq. and Luther Holden, Esq., 'Lancet,' 1847, vol. i, p. 441.—The Nature and Treatment of Erysipelas: being the subject for the Jacksonian Prize for the year 1849, by Peter Hinckes Bird, Esq., M.S. Library of the Coll. of Surgeons.

smallpox only, as much uninfluenced by other diseases as the same number of cases would be if taken from private and parochial practice, where the accommodation for the sick is often anything but favorable for their recovery, except in selected cases, which of course would not apply to the community at large, as this question ought to be applied, any more than hospital cases, so that when the remaining evils between hospital and private and parochial practice have been relatively balanced, the results would be perhaps nearly the same.

Viewing the question, then, as it actually occurs in practice, and to simplify, and enable me to make my report on this large number of patients, 5982, as clear and concise as appeared to me to be possible, they have been arranged annually for sixteen years, on one systematic plan, and from these annual and other tables, altogether 87 tables, the whole have been combined, and finally formed into a series of six tables, comprising, it is hoped, most of the principal points of interest in the inquiry.

Of the series, the first table shows the number of patients admitted at the hospital in each year, distinguishing males from females, whether vaccinated or otherwise, and gives the outline of the disease under which each was suffering. It includes 185 cases of febrile diseases, principally eruptive, but not variolous, although sent to the hospital as such, and furnishes the result of the whole, with the rate per cent. of mortality.

The second table gives an analysis of all the cases of smallpox, viz. 5797, classed under nine different headings, the form of the disease in each case, and the result, with the rate per cent. of mortality under each division.

The third table gives the ages of the unprotected patients, and the rate per cent. of mortality, calculated at different periods of life, for every five years up to thirty, and every ten years afterwards.

The fourth table further exhibits, separately, the leading particulars of 3094 cases of smallpox after vaccination, showing, from a careful examination of each patient, the number and character of the vaccine cicatrices, the form of

the variolous disease, and the result, with the rate per cent. of mortality from smallpox, after deducting the cases of superadded disease. It also shows the rate of mortality, from smallpox, in patients having one vaccine cicatrix, particularising whether good or indifferent, and the average; as well as with two, three, four, or more cicatrices; and of those reported to have been vaccinated, but who were without any cicatrix.

The fifth table shows, in quinquennial periods, the ages of the vaccinated patients when attacked by smallpox; *where* they were vaccinated, and the rate of mortality.

And the sixth table states, in periods of five years, the ages of the patients at the time they were vaccinated, who have subsequently been admitted with smallpox at the Smallpox Hospital.

There is probably no subject in the whole range of medical science to which the numerical method of investigation is more applicable than to the results obtained, and the effect produced on life, by vaccination. At the present day it is unnecessary for me to dwell on its advantages over other modes of inquiry; it has, fortunately for the advancement of our art, taken the place in a great measure of hypothesis and mere opinion, which was of but little value, for the most part, on subjects of inquiry like the present, unsupported by any documentary evidence, compared with the exactness we arrive at by arranging and classifying a large number of kindred cases, and drawing our deductions from the whole. Much the same impression might have been and doubtless was often left on a careful, observant mind, but then other persons could not have been equally satisfied that the deductions drawn were the correct ones, as they can be when the cases and the particulars are placed numerically before them, and from which each inquirer can form his own opinions. The tables which I have thus arranged, and now present to the Society, contain information which has occupied a portion of every day in collecting for sixteen years, and which may be combined and studied in a variety of ways; on some of the leading features of each table I will proceed to offer a few comments, taking them in the order of succession.

TABLE I.

Summary and General Classification of the Patients admitted annually at the Smallpox and Vaccination Hospital, London, during a Period of Sixteen Years, from 1836 *to* 1851, *inclusive.*

YEARS.	Number of patients.			Natural smallpox.		Smallpox after smallpox.		Smallpox after vaccination, with and without cicatrices.		Smallpox after vaccination or inoculation.		Febrile diseases, principally eruptive, but not variolous, although sent to hospital as such.		RESULTS.					
															Died.				
	Males.	Females.	Total.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Discharged.	Males.	Females.	Total.	Affected by superadded disease.	Rate per cent. of mortality.
1836	215	114	329	185	70	1	–	131	12	–	–	12	2	245	59	25	84	4	25·53
1837	156	95	251	142	44	–	–	97	1	–	–	12	1	205	31	15	46	5	18·32
1838	424	288	712	387	155	–	–	306	33	–	–	19	–	524	117	71	188	30	26·40
1839	102	53	155	61	22	–	–	83	4	1	1	10	–	128	16	11	27	1	17·41
1840	186	141	327	189	87	3	–	124	8	–	–	11	–	232	52	43	95	5	29·05
1841	209	148	357	189	64	2	1	151	10	–	–	15	–	282	51	24	75	5	21·00
1842	91	64	155	77	30	2	–	62	4	–	–	14	–	121	23	11	34	4	21·93
1843	92	68	160	77	27	2	–	69	–	–	–	12	–	133	21	6	27	3	16·87
1844	391	256	647	302	117	4	3	337	50	–	–	4	1	496	87	64	151	18	23·33
1845	240	144	384	137	63	4	–	227	15	–	–	16	1	305	54	25	79	3	20·57
1846	96	56	152	57	22	2	–	88	7	–	–	5	–	123	17	12	29	2	19·07
1847	270	191	461	167	50	8	3	275	28	–	–	11	–	380	49	32	81	10	17·57
1848	449	247	696	254	103	4	2	428	63	–	–	10	–	528	99	69	168	39	24·13
1849	123	77	200	71	22	4	–	115	11	–	–	10	–	167	22	11	33	5	16·50
1850	199	115	314	129	42	2	–	174	16	–	–	9	–	256	43	15	58	3	18·47
1851	438	244	682	230	78	9	–	427	26	1	–	15	–	578	64	40	104	9	15·24
Totals....	3681	2301	5982	2654	996	47	9	3094	268	2	1	185	5	4703	805	474	1279	146	21·38

TABLE I.—The numbers admitted each year will be found to have fluctuated considerably, of course, in an hospital devoted to the reception of patients suffering from a disease liable to great variation from its epidemic character; so much so that during some years the admissions have been four or five times more numerous than in others. The total difference in the admission of males and females has been as three to two, three males to two females; the rate of mortality has been nearly the same, less by 1 per cent. only in females than in males. The average annual mortality has been 21 per cent., ranging between a minimum of 15 and a maximum of 29. And it will be found generally to have been greater in epidemic times, owing partly to the greater severity of the disease, and partly, probably, to the evil influence of collecting together, in a space too limited for them, a large number of sick persons. This will be rendered visible by observing the diminished mortality which has already taken place, during a severe epidemic since the new hospital at Highgate Hill has been in use, which is placed in a more healthy situation than the old hospital at Battle Bridge was, and where the space for patients is much larger, upwards of 2000 cubic feet being allowed for each. The general sanitary arrangements are also much better. The new hospital came into use in July, 1850.[1] It ought, however, to be stated that on three different occasions, during the epidemics of 1838-44-48, when it was impossible to receive, for want of room, all the applicants for admission, and in order to afford as much assistance to the public as possible, severe cases only were admitted, and many mild cases, after vaccination, recommended to find accommodation elsewhere. In this way the mortality at the hospital was necessarily increased, rendered greater per cent. than it would have been had all the applicants been admitted indiscriminately. It has besides, fre-

[1] After having given considerable attention to the subject for several years, I have observed that the cubical space allowed for patients with smallpox, and like diseases, such as typhus, scarlatina, &c., should not be less than 2000 cubic feet for each patient.

quently come to my knowledge at other times, that a severe case has been sent to the hospital, whilst a mild case or two, in the same house, has been kept at home. This is only what might be expected, but it helps to account for the increased mortality there may be per cent. in hospitals from any given disease, over the mortality per cent. from the same disease amongst the public at large in private houses.

185 cases of febrile diseases, not variolous, have been admitted in the sixteen years, and about twice as many were seen on their arrival by the medical officers of the hospital prior to admission, and were not received; but as this does not in any way alter the order of frequency in which errors of diagnosis occurred with regard to the several mistaken diseases, it will probably be interesting to the Society to know the diseases which have been most commonly mistaken for smallpox. When arranged, they come under 21 different heads, but, as may be supposed, some diseases have misled much more frequently than others. Of the 185 cases, 50 were measles, 33 lichen febrilis, 30 varicella vera, 27 fever, making 140 of the 185. The next disease in order of frequency is urticaria, of which there were 8 cases. It will, therefore, be seen that measles, lichen, varicella, and fever, have led to mistakes much more frequently than any other diseases. Of the cases of fever, a few arose, in my opinion, from the infection of variola in vaccinated individuals, which terminated in two or three days with the initiatory fever, but without the formation of the characteristic variolous eruption.

TABLE II.

Analysis of the Cases of Smallpox admitted at the Smallpox and Vaccination Hospital, London, from 1836 *to* 1851, *inclusive.*

Patients admitted with Small Pox.	Number of Patients.	Forms of the Disease.												Results.			
		Eruption regular or unmodified.						Eruption irregular or modified.									
		Confluent.	Died.	Semi-confluent.	Died.	Distinct.	Died.	Confluent.	Died.	Semi-confluent.	Died.	Distinct and varicelloid.	Died.	Discharged.	Died.	Died, affected by superadded disease.	Ratio per cent. of Mortality from Smallpox, after deducting entirely the cases affected by superadded disease.
1. Unprotected	2654	1821	936	597	51	167	8	17	1	17	–	35	–	1658	996	81	35·55
2. After natural Smallpox	14	3	—	2	–	—		3	–	3	–	3	–	14	—	—	—
3. After inoculated Smallpox	27	10	5	2	1	—	–	4	1	4	–	7	–	20	7	1	23·07
4. After Vaccination and Smallpox	5	2	2	—	–	—	–	1	–	—	–	2	–	3	2	—	Too limited for averages.
5. After Vaccination, or Inoculation, and Smallpox	1	1	—	—	–	—	–	—	–	—	–	—	–	1	—	—	
6. After Vaccination or Inoculation	2	2	1	—	–	—	–	—	–	—	–	—	–	1	1	—	
7. After Vaccination, with cicatrix or cicatrices	2787	428	153	232	6	89	–	486	24	416	3	1136	5	2596	191	47	5·25
8. Stated to have been vaccinated, but having no cicatrix	290	138	67	33	1	16	1	42	5	20	–	41	–	216	74	14	21·73
9. Stated to have been vaccinated, but particulars of cicatrix not recorded	17	4	2	2	1	3	–	1	–	—	–	7	–	14	3	2	6·66
Total	5797	2409	1166	868	60	275	9	554	31	460	3	1231	5	4523	1274	145	19·97

TABLE II.—Of the 5797 cases of smallpox, 2654, or 45 per cent., were unprotected; 47 cases, or less than 1 per cent., were after a previous attack of smallpox, or smallpox inoculation; 3094 cases, or 53 per cent., were after vaccination.

69 of the unprotected patients had the eruption modified as it is by vaccination, although it could not be ascertained that any of these patients had ever undergone vaccination. They were examples of mild natural smallpox, such as have no doubt occurred at all periods to a few favoured individuals, and in which, fortunately for the objects attacked, the disease leaves no trace behind. Some of the cases occurred in little children, whose mothers were at the hospital with them, and by whom it was perfectly well known no vaccination had ever been attempted. After making allowance in the unprotected for those who died affected by superadded disease, which was about 2 per cent., there died $35\frac{1}{2}$ per cent. of smallpox.

14 patients had smallpox after a previous attack of natural smallpox, and not one died. 27 patients had smallpox after having been some years before inoculated for smallpox, and they died at the rate of 23 per cent.

2787 bore marks of having been previously vaccinated; and after deducting the cases affected by superadded disease, which amounted, as in the unprotected, to 2 per cent., there remained a mortality of $5\frac{1}{4}$ per cent. from smallpox.

TABLE III.

Ages of the unprotected Patients admitted with Smallpox at the Smallpox and Vaccination Hospital, London, from 1836 *to* 1851 *inclusive, with the rate per cent. of Mortality, calculated at different periods of life.*

Date. 1836—1851.	Age in Years.												Totals.
	0—5	5—10	10—15	15—20	20—25	25—30	30—40	40—50	50—60	60—70	70—80	80—90	
Patients . .	356	334	270	571	669	270	154	18	8	2	1	1	2654
Deaths . .	181	91	62	154	274	124	89	13	5	1	1	1	996
Per centage of deaths .	50	27	23	26	40	45	57	69		75			37

TABLE III.—Natural smallpox will be seen to be a most fatal disease at all periods of life; the most so in infancy, and advanced life; the least so from 10 to 15 years of age; under 5 years it is 50 per cent.; still greater, however, under two years; the mortality after the age of 20 rises suddenly, and increases gradually; at 30 it exceeds the mortality of infancy, and after 60 hardly any escape.

TABLE IV.

Analysis of the Cases of Smallpox after Vaccination, admitted at the Smallpox and Vaccination Hospital, London, from 1836 *to* 1851, *inclusive, showing, from a careful examination of the Cicatrices, the relative amount of security given by the number of vesicles produced at Vaccination; and, judging from the character of the Cicatrices, the probable state of activity and efficacy of the Lymph used for Vaccination.*

Patients admitted with Small Pox.	Number of Patients.	Character of the Cicatrices.	Cases.	Forms of the Disease.														Results.				
				Eruption unmodified in 945 cases.							Eruption modified in 2149 cases.											
				Confluent.	Died.	Semi-confluent.	Died.	Distinct.	Died.	Number of unmodified cases.	Confluent.	Died.	Semi-confluent.	Died.	Distinct and varicelloid.	Died.	Number of modified cases.	Discharged.	Died.	Died affected by superadded disease.	Rate per cent. of mortality from smallpox, after deducting entirely the cases affected by superadded disease.	
1. Having one vaccine cicatrix	1357	good	768	107	34	72	2	30	–	209	131	5	133	1	295	2	559	724	44	12	4·23	7·57
		indifferent	589	168	68	57	1	26	–	251	110	11	80	–	148	1	338	508	81	12	11·95	
2. „ two vaccine cicatrices	888	good	608	66	20	44	2	12	–	122	115	4	91	–	280	1	486	581	27	11	2·68	4·13
		indifferent	280	51	21	31	1	9	–	91	43	3	40	1	106	–	189	254	26	6	7·29	
3. „ three vaccine cicatrices	274	good	187	13	5	12	–	6	–	31	22	1	34	–	100	1	156	180	7	4	1·63	1·85
		indifferent	87	7	3	7	–	5	–	19	17	–	11	–	40	–	68	84	3	1	2·32	
4. „ four or more vaccine cicatrices	268	good	202	10	2	9	–	1	–	20	38	–	22	–	122	–	182	200	2	–	0·99	0·74
		indifferent	66	6	—	—	–	—	–	6	10	–	5	1	45	–	60	65	1	1	0·00	
5. Stated to have been vaccinated, but having no cicatrix	290		290	138	67	33	1	16	1	187	42	5	20	–	41	–	103	216	74	14	21·73	
6. Stated to have been vaccinated, but particulars of cicatrix not recorded	17		17	4	2	2	1	3	–	9	1	–	—	–	7	–	8	14	3	2	6·66	
	3094		3094	570	222	267	8	108	1	945	529	29	436	3	1184	5	2149	2826	268	63	6·76	

TABLE IV.—3094 patients with smallpox reported themselves to have been vaccinated at some period of their lives. Patients were never entered in the register as vaccinated unless the account of the vaccination was a tolerably clear one, either from the patient's own recollection, or the account received of it from his or her friends,—their belief that it had taken effect properly, and their trusting to it as their protection against smallpox. The only exceptions to this statement were—1st. Those who had the usual vaccine cicatrices on their arms, but knew nothing of having been vaccinated. 2d. Patients who were admitted while under vaccination; they were considered, at whatever stage the vaccination had arrived, as unprotected, and entered accordingly. Of the whole 3094 cases after vaccination, with and without cicatrices, there died, after deducting the cases of superadded disease, $6\frac{3}{4}$ per cent. 945 of the vaccinated cases were unmodified, and 2149 modified. Of the 945 unmodified cases, there died 231, or 24 per cent., by which it will be observed that there is a difference of one third in the mortality between these cases and the unprotected cases; therefore, although the eruption was not recorded in the register as modified or mitigated, the constitution must have received a protective influence, as regards fatality, to the amount of one third, or else there would not have been this difference in the number of deaths. Many cases were not entered as modified, though in my opinion at the time they would have been more correctly so entered; that opinion is sustained by the mortality, which would have been greater but for the disease having been influenced by vaccination.

1357 patients had one vaccine cicatrix, and of these there died, with a good cicatrix, $4\frac{1}{4}$ per cent.; with an indifferent cicatrix, just upon 12 per cent., the average being $7\frac{1}{2}$.[1]

[1] A good vaccine cicatrix may be described, as distinct, foveated, dotted or indented, in some instances radiated, and having a well, or tolerably well, defined edge.

An indifferent cicatrix, as, indistinct, smooth, without indentation, and with an irregular and ill-defined edge.

888 patients had two cicatrices, and there died, with good cicatrices, $2\frac{1}{4}$ per cent.; with indifferent cicatrices, $7\frac{1}{4}$ per cent.; the average being a little over 4 per cent.

274 patients had three cicatrices, the mortality being with good cicatrices $1\frac{1}{2}$ per cent.: with indifferent cicatrices $2\frac{1}{4}$ per cent.; average $1\frac{3}{4}$ per cent.

268 patients had four or more cicatrices, and there died with good cicatrices just under 1 per cent.; with indifferent cicatrices none, the average being only $\frac{3}{4}$ of 1 per cent. There is a difference in the last statement between the mortality of those having good and indifferent cicatrices that does not accord with the previous results; but all candid minds will readily be convinced that the average would have been preserved had the numbers been greater to calculate from, for when all the numbers are taken together, there died, with good cicatrices, 3·04 per cent.; with indifferent cicatrices, 9·77 per cent. Then, again, as regards the number of cicatrices, there died with one and two cicatrices, 6·21 per cent.; with three, four, and more cicatrices, 1·30 per cent.

The danger from smallpox arises in the great majority of instances, solely from the quantity of eruption—from the extensive interruption and destruction of the functions of the skin produced by the pustules, in much the same way as from a severe burn or scald. Death may take place, and commonly does take place, without any perceptible disease of the internal organs essential to life, excepting the pustules on the air-passages, and congestion of the lungs, the latter being produced by the additional duty thrown upon them, as occurs whenever the healthy functions of the skin are interrupted. Knowing this,—that the danger arises principally from the quantity of eruption,—we may examine the question in another light. By referring to Table IV, it will be seen that, of 2245 patients, with one and two vaccine cicatrices, 392, or $17\frac{1}{2}$ per cent., had smallpox in a confluent unmodified form. Whilst of 542 patients with 3, 4, or more cicatrices, 36, or $6\frac{1}{2}$ per cent. only, had the confluent unmodified disease. Again, 1765 patients had *good* cicatrices, of whom 196, or 11 per cent., had the confluent un-

modified disease; whilst of 1022 with *indifferent* cicatrices, 232, or 22 per cent., just double the per centage of the above, had it in the unmodified form. Test the question in which way soever we will, the result is in favour of producing four vesicles at least at vaccination, with lymph that leaves good permanent cicatrices.

290 patients reported themselves vaccinated, but had no cicatrices, and they died at the rate of $21\frac{3}{4}$ per cent., which teaches us the important fact, that the mortality from smallpox is more than four times as great in persons who, though believing themselves protected by vaccination, are without cicatrices, as it is in those who bear vaccine cicatrices. Practically, this is a point of very great importance, and persons so circumstanced as to have no cicatrix, should be re-vaccinated, if young, upon attaining puberty, or at any subsequent period, without delay. 17 patients were entered as vaccinated, but the particulars of the cicatrices have been omitted in the register. These cases occurred in 1836-7-8. The collected number ought to have been rather larger. In making the tables, the cases were arranged yearly from 1836 to 1851, and one or two patients some years were found to have had the particulars about the cicatrix omitted. The numbers being unimportant, they were incorporated, for the sake of simplicity, with the list of cases without cicatrices; but when so many as 17 were found in 3 years, it was thought to be better to give them a separate heading. The total number of cases, however, so added to the list without cicatrices, did not exceed 20, and, so far as it goes, makes the per centage of deaths of the class without cicatrices rather less than it ought to have been.

TABLE V.

Ages of the Patients admitted with Smallpox after Vaccination, at the Smallpox and Vaccination Hospital, London, from 1836 *to* 1851 *inclusive; and where they were vaccinated, so far as could be ascertained.*

Where Vaccinated.	Number of Patients.	Ages of the Patients.																		Results.			
		0—5		5—10		10—15		15—20		20—25		25—30		30—35		35—40		40-50		50-60					
		Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Discharged.	Died.	Died affected by superadded disease.	Rate per cent. of mortality from smallpox, after deducting entirely the cases affected by superadded disease.
Vaccinated at the stations of the National Vaccine Establishment, or at the Smallpox and Vaccination Hospital, or at the stations of the Royal Jennerian Society in London .	155	2	–	12	3	33	4	68	4	24	—	11	1	3	1	2	—	—	—	–	–	142	13	4	5·96
Vaccinated in the Metropolis, but not at the Public Vaccine Institutions, so far as could be ascertained	370	5	2	29	3	76	1	135	7	77	5	26	3	13	1	3	—	6	1	–	–	347	23	5	4·93
Vaccinated in the Provinces of England and Wales	1987	–	–	8	1	62	3	528	29	753	72	363	34	153	17	74	18	44	7	2	1	1805	182	42	7·19
Vaccinated in Scotland . . .	79	–	–	—	–	—	–	16	1	36	2	15	2	7	—	4	—	1	–	–	–	74	5	2	3·89
„ in Ireland . . .	35	–	–	—	–	8	–	8	2	13	—	2	—	2	—	2	—	—	–	–	–	33	2	—	5·71
„ in Foreign Countries	279	–	–	2	–	6	1	47	1	103	6	80	13	19	1	16	2	6	1	–	–	254	25	6	6·95
Not known where Vaccinated .	189	–	–	5	–	21	1	64	5	52	8	29	2	13	1	1	—	4	1	–	–	171	18	4	7·56
Totals . . .	3094	7	2	56	7	206	10	866	49	1058	93	526	55	210	21	102	20	61	10	2	1	2826	268	63	6·76

TABLE V. But few patients under 10 years of age have been received with smallpox after vaccination. After 10 years the numbers begin to increase considerably, and the largest admitted are for the decennial period from the age of 15 to 25; and, although progressively diminishing, they continue rather large up to 30; from 30 to 35, they are nearly the same as from 10 to 15; but as in the unprotected, at this period of life the mortality is doubled, showing the cause to be, probably, as much or more depending on age and its concomitants, as on other circumstances. In still further advanced life, the rate of mortality will be seen to increase also, as in the unprotected state; but this tendency may be in a considerable degree counteracted there is but little doubt, by giving more attention than has hitherto generally been given to the perfection of the process of vaccination.

155 patients are reported to have had smallpox after having been vaccinated at the public vaccine institutions of London.

370 had been vaccinated in the metropolis, but not at the public vaccine institutions, so far as could be ascertained.

1987 patients were stated to have been vaccinated in the provinces of England and Wales.

79 in Scotland.

35 in Ireland. Only two a year. This is a very remarkable circumstance, when we bear in mind the large mass of Irish population resident in this metropolis, besides those here for a short time every year.

279 had been vaccinated in foreign countries; this number also includes a few, but very few, vaccinated in the British colonies.

189 did not know where they had been vaccinated: they were English, and principally country people.

The question, then, as regards the place where the vaccinations were performed, of the patients subsequently admitted with smallpox at the hospital, stands, collectively, per cent., thus:

Vaccinated at	the Public Vaccine Institutions of London	5 per cent.
„	in the Metropolis, but not at the Vaccine Institutions, so far as could be ascertained . . .	11 „
„	in the Provinces of England and Wales	64 „
„	in Scotland	2 „
„	in Ireland	1 „
„	in Foreign countries . . .	9 „
Not known were vaccinated		6 „
		98 „

The remaining 2 per cent. are made up of fractions of the above.

TABLE VI.

Ages of the Patients at the time they were Vaccinated, who were subsequently admitted with Smallpox, at the Smallpox and Vaccination Hospital, London, from 1836 *to* 1851, *inclusive.*

Date of Admission with Small Pox. 1836—1851.	Ages in Years when Vaccinated.										Totals.
	0—5	5—10	10—15	15—20	20—25	25—30	30—40	40—50	50—60	Ages when vaccinated not stated.	
Patients	2794	101	64	17	6	—	—	—	1	111	3094

TABLE VI.—Of the 3094 patients admitted with smallpox after vaccination, 2794, or 90 per cent., had been vaccinated when under five years of age.

My opportunities of examining, with regard to previous vaccination, the foreigners admitted with smallpox at the hospital, and comparing them with each other, and with the same class of persons in this country, have led me to the conclusion that vaccination is performed in the best manner, generally, by the Danes, Swedes and Norwegians, and Germans, judging them by the standard shown in Table IV to afford the most effectual security: then come the Italians, and, from the few I have seen, the Spaniards; then the Scotch; then the Irish; and lastly, the English and French. The French who have been admitted as patients at the Smallpox Hospital, appear to have been very indifferently vaccinated; they have been but few, it is true, and these few may have been of the few badly vaccinated in France.

It will be observed, that two thirds of the whole number admitted with smallpox after vaccination had been vaccinated in the provinces of England and Wales. By 'provinces,' meaning the whole of England and Wales, except London, and the area included in the metropolitan district. I am most anxious to draw the attention of my professional brethren in the country to the above fact; and amongst that enlightened community, the feeling can scarcely fail to be otherwise than general, that there must exist some grave and lamentable evils (more especially affecting the humbler classes) connected with the circumstances under which vaccinations in country districts are performed. The details recorded in this paper may be urged as calling upon those of our provincial brethren whose position and ability give them influence, to lend their aid in tracing the evil to its root, and using their endeavours to remove a stigma, which, whatever may be its origin, is too likely to be shared by themselves with less skilful, less conscientious, and less scrupulous men. Some reasons may, perhaps, be assigned for a large number of persons in country districts being imperfectly vaccinated,

but there can be no justifiable reason why the rural inhabitants of England and Wales should be far less well vaccinated than are the rural inhabitants of Denmark, Sweden, and Prussia. That they are so, I know to be a fact, from the opportunities presented to me of observing large numbers of Germans more especially, who, from the efficient way in which they had been vaccinated, have had smallpox usually in the light varicelloid form. The subject is a delicate one to allude to—to point out defects is always painful, but the vital importance of the subject as affecting the health and lives of thousands, will be my excuse for the remarks now made, and for further calling attention to Table v, and the collected per centages at page 379. When so many of the lives of our patients are at stake, to say nothing of minor, though still grave evils, that may be prevented by greater care and better management,—it becomes a positive duty to express our opinions as strongly as may be consistent under the circumstances; to make at least an appeal to the profession, which will no doubt be promptly responded to by a more careful performance of an operation, which, though of minor importance in itself as a surgical operation, is of most serious importance in its consequences, as affecting health and life. If the great discovery of Dr. Jenner can avert, as I have shown it can when effectually carried out, the serious results of this terrible disease, it is surely desirable to be aware of any facts which might point out the means, and the necessity of performing the operation in a more complete manner. With good lymph, and the observance of all proper precautions, an expert vaccinator should not fail of success in his attempts to vaccinate above once in 150 times; yet a large number of those who take upon themselves the duty, think they do very well if they succeed, however imperfectly, five times out of six. Patients often present themselves with smallpox at the hospital, who state they have been cut 5, 6, 8 times, or more, for cowpox without effect. This is a great evil. It would happen but rarely in careful hands. Such persons think it is of no use having the operation tried again, that it will not take effect if they

do, and ultimately they are attacked by smallpox, and perhaps die; whereas, had they fallen into the hands of a good vaccinator, their lives would most likely have been saved. Instances of the above kind are of frequent occurrence at the Smallpox Hospital.

In December, 1836, a woman, æt. 54, and her son, æt. 15 years, were admitted with smallpox, and died in the course of five days: the daughter, a young woman, died of smallpox at their house in Hull Street, City Road, the day before the mother died at the hospital. All these poor people had been vaccinated three years previously, at a parish vaccination, at a village church in Suffolk, and bore marks of the vaccination. Now, knowing what we do of the protective powers of vaccination, it is almost impossible to suppose otherwise than that these poor people had been vaccinated with improper lymph. They, however, believed they had been efficiently vaccinated, and trusted to it for their security against smallpox. These three lives fell a sacrifice, most likely, to mere carelessness.

The mortality alone, severe as it is, between the indifferently vaccinated, and the well vaccinated patients, is not the only evil result to be regretted of bad vaccination. Proportionate to the mortality has been the severity of the disease in those who escaped death; damaged health, and disfigurement for life is frequently the lot of those who have smallpox after having been vaccinated imperfectly in but one place; thus bringing on vaccination discredit, which is in no way due to it intrinsically, but is owing solely, with but very few exceptions indeed, to the want of proper knowledge of the subject, and of the necessary care with which the operation has been conducted.

Great judgment and caution should be used in the selection of vaccine lymph; in this lies one of the principal causes of failure in vaccinating, and subsequent insecurity of the individual, even when the vaccination does take effect. This advice is offered after seventeen years of constant practice of vaccination, and after the experience acquired by vaccinating between forty and fifty thousand persons.

Lymph for use is in its best state on the seventh day of the progress of the vesicle it is taken from—the day week from the vaccination. It should be taken when the vesicles are plump, and just before the formation of the areola. Under no circumstances should it be taken for use later than twenty-four hours after the areola has begun to form. If this rule were invariably observed, there would be, as I believe, but very few cases of severe smallpox after vaccination. At this stage of the progress of the vesicle the lymph is in the state most certain of taking proper effect, and of leaving the best cicatrices, indicative of its efficacy, a result shown in the accompanying tables to be of the greatest importance to individual security, in case of smallpox occurring in after life. A serious error in vaccinating is the use of blunt lancets—lancets unsuitable for other purposes. It is impossible to have a lancet too sharp for vaccinating, the sharper the lancet, the more perfect the success of the operation, even in good hands.

Mode of conveying lymph.—A convenient way, in practice, of conveying lymph about for use, is in stoppered bottles, the stopper on which the lymph is placed, and kept fluid, being ground flat, and projecting into the bottle. The lymph ought not, however, to be kept in this way above twenty-four hours in warm weather, or forty-eight in cold. Like all other moist animal matter, it soon begins to undergo chemical changes, which render it unfit for use. When it has become putrid, or even putrescent, it will produce the fatal results which are well known to follow inoculation with decaying animal matter. Much the same remarks apply, *mutatis mutandis*, to the lymph preserved in capillary tubes, as to that preserved in bottles.

Mode of vaccinating.—The arm to be operated on should be firmly grasped by the left hand of the operator, so as to make the skin tense, then, the lancet being already charged, with the right hand the lymph should be introduced by a puncture of a valvular shape, from above downwards, so managed that the lymph at each puncture may gravitate into the wound. In this way the lymph may be introduced

in five punctures,—the number I recommend,—from half to three fourths of an inch apart, without recharging the lancet; the skin being kept tight all the while, until the lymph has been introduced, care being taken that the punctures are not bruised, as too frequently happens by undue use of the lancet. It then matters not how much the wounds may bleed, as the bleeding will not interfere with the success of the operation.

Mode of preserving lymph.—By far the best way of preserving vaccine lymph, and also of transmitting it to a distance, is on ivory points. They should be *well* charged, not simply touched; the quantity allowed to dry, should be about equal in its wet state to half a drop; when the charged points are required for use, they should be lightly dipped in water, and placed for a few minutes on the edge of a book, so that the lymph may have time to become soft, much as it was when taken from the vesicle. Then, after making a puncture with a lancet, the ivory point may be inserted into the arm, and kept there with the thumb for a short time; or the moistened lymph may be scraped off the point, and at once inserted with the lancet. Several little niceties are required in vaccinating with preserved lymph, which are not, generally, so much attended to as they deserve to be, in order to ensure perfect success.

Re-vaccination.—For many years past I have practised re-vaccination extensively on persons applying for the purpose at the vaccination room; on the servants and nurses of the hospital, on persons coming to visit their friends, patients in the hospital, and, lately, on the numerous workmen employed in building the new hospital. The effect produced by re-vaccination sixteen or seventeen years ago was, with some few exceptions, nothing more than a little irritation, or at most an abortive vesicle with irregular areola. But, during the last three or four years, I have seen a great many persons, on whose arms the vesicles produced by re-vaccination have been quite, or nearly perfect, even on those who bore good cicatrices from the first vaccination. I have always recommended revaccination after puberty,

principally for this reason, it gives those who have been indifferently vaccinated in infancy another chance of being protected. Probably it does not afford the same amount of protection that the first vaccination, well performed, does. The great object to aim at is to vaccinate *well* in infancy, this should be looked upon as the sheet-anchor; and, therefore, a careless vaccination should be deprecated at all times, practised under the belief that if it fails to take effect properly, it will be of no consequence, as the operation can be repeated. By such proceeding the vaccination often takes effect badly, and will never afterwards take effect properly, and yet the individual may take smallpox severely. This should be viewed as, unfortunately, one of the imperfections in the practice of vaccination, but the knowledge of it teaches us the paramount importance of paying the strictest attention to the mode of performing the *first* vaccination.

In 1838 smallpox attacked the children in the Deaf and Dumb Asylum, and one or two a week, for three or four weeks, sickened with it, when Dr. Babington,[1] physician to the Asylum, requested me to re-vaccinate the whole of the inmates, about 260. I did so. Four days afterwards another child was attacked, who had received the infection of smallpox before he was re-vaccinated, but from this date the disease was arrested in the establishment. Some months subsequently, a servant, who had come fresh into the asylum, had smallpox, but the disease on this occasion did not spread to the other inmates.

For upwards of seventeen years of my connection with the Smallpox Hospital, not one of the servants or nurses of the hospital has been attacked by smallpox, although vaccination has been the only protection of many of them; but I have always re-vaccinated them on their first coming to live at the hospital.

[1] For an account of two similar occurrences and results, see a paper entitled, Cases of Smallpox which occurred in the Asylum for the Deaf and Dumb, with an account of the Re-vaccination of all the Children in that establishment, by Dr. Babington, 'Guy's Hospital Reports,' vol. i, p. 159.

On rebuilding the hospital lately, a large number of workmen were employed for several months after the arrival of the patients, most of these workmen consented to be re-vaccinated, two only were attacked by smallpox, but *they* were amongst the few who were *not re-vaccinated.*

The inferences deducible from the foregoing facts and statements are:

1. That natural smallpox destroys about one third of all whom it attacks.

2. That smallpox after smallpox is of comparatively rare occurrence in a population who owe their protection in large numbers to having gone through the natural, or inoculated disease. That a second attack of natural smallpox, as well as being rare, is probably not often fatal, and that protection seems to be the law. That after inoculated smallpox, an attack of smallpox has more frequently led to fatal results, but there is reason to presume, that the virus used for inoculation, like a great deal of the lymph used at the present day for vaccination, was often taken at too advanced a stage of the disease, and thus did not afford the full measure of protection it was capable of affording, if taken at a proper time. That when the disease does occur, it is sometimes modified, as it is after vaccination, and that the modification presents the same characters. That second attacks of smallpox are further presumed to be of very rare occurrence, as there is no instance recorded of a person admitted with smallpox a second time at the Smallpox Hospital, although the hospital has now been founded 107 years. That, as upwards of 20 different diseases are known by the author to have been mistaken for smallpox during the last sixteen years, and sent as such to the Smallpox Hospital, it may be fairly assumed that many of the cases reported amongst the public, during the last quarter of a century, as second attacks of smallpox, have been either in the primary or secondary instance, no smallpox at all. That smallpox does, however, occur a second time there can be no reasonable doubt, and one of the circumstances that seems to predispose the constitution to receive a second attack of the disease is, as after vaccination,

exposure for a time to great change of climate, either hot or cold.

3. That vaccination performed in infancy affords almost complete protection against the fatality of smallpox to the period of puberty. That a variety of circumstances conspire to make it almost impossible to ascertain exactly in what proportion to the vaccinated, cases of smallpox do subsequently occur, or might occur, if all persons lived to an advanced age; in illustration of which it may be stated that a woman, 83 years of age, was admitted at the hospital in 1844, with severe confluent natural smallpox, of which she died, who had nursed her own children, and her grandchildren with the disease, and had otherwise been exposed often to variolous infection, but never took it before. That of the small proportion to the great mass protected by vaccination, who do unfortunately take smallpox in after life, of those who have been vaccinated indifferently in but one place, about 12 per cent. die; but that of those who have been well vaccinated in four places or more, the proportion of deaths is less than 1 per cent.; so that by careful management the protection to life may be rendered all but perfect.

4. That as a matter of safety, it is well for all persons who were vaccinated in infancy to be revaccinated at puberty, this measure being more especially requisite for those who were either indifferently or doubtfully vaccinated in infancy, and still more especially necessary for those who though vaccinated, have no cicatrix remaining. Finally, as a matter of precaution, it is desirable that all persons should be revaccinated, on smallpox existing in the house where they are residing; a precaution, however, that will cease to be necessary to advise when all persons have the benefit of proper and efficient vaccination.

A circumstance has come to my knowledge in prosecuting this inquiry, which, in conclusion, I beg to submit to the Society. And although its bearing is not so rigidly precise as the rest of this communication, the point it conveys is of an important character, substantially true I believe, and

proper to be brought forward on this occasion, as showing, on a large scale, the protective powers of vaccination. Already it has been stated that in sixteen years, 155 patients, with and without vaccine cicatrices, or about 9 a year, have been admitted with smallpox at the Smallpox Hospital, who were reported to have been vaccinated at the Public Vaccine Institutions of London. In an Appendix to the Report from the Select Committee of the House of Commons on the Vaccine Board, in 1833,[1] there are some official returns for the previous five years, of the numbers vaccinated annually at the different stations of the National Vaccine establishment, at the Smallpox and Vaccination Hospital, and at the stations of the Royal Jennerian Society in London, which, together, will be found to amount to 102,114; this, divided by 5, gives an average of 20,422 as vaccinated yearly. About the same numbers had been vaccinated at these institutions for several years before the returns were made, were continued, somewhat increased, for several years later, until after the time of passing the Vaccination Act in 1840, and, with perhaps some slight diminution of late years, have been continued ever since. These persons so vaccinated were for the most part poor persons, and also for the most part stationary, living too in a town where smallpox is never absent, and in case of being attacked by it, likely to apply for admission at the Smallpox Hospital, the only place in London for the reception of smallpox patients; yet the numbers admitted with smallpox, who had been vaccinated at these public vaccine institutions, have amounted but to a fraction over 9 a year, or 1 to 2108 vaccinated; and the deaths from smallpox but to 1 in 36,305 vaccinated.

This may be truly designated one of the triumphs of medical science; a result more favorable and gratifying could hardly have found a place in the most sanguine and philanthropic hopes of the humane and illustrious Jenner.

[1] Report from the Select Committee on the Vaccine Board, with the Minutes of Evidence, and an Appendix, ordered by the House of Commons to be printed, 28th August, 1833, Appendix A, B, C, pp. 137, 141, 149.

OBSERVATIONS ON THE STATE OF THE BLOOD AND THE BLOOD-VESSELS IN INFLAMMATION.

BY

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It has not unfrequently been objected to the results obtained from microscopical observations on the web of the frog in the inflamed state, that we cannot safely argue from them as to the nature of the inflammatory process in man.

This, however, like many other general objections, is well or ill founded according to the sense in which the terms are employed. If in the objector's mind, the word "inflammation" conjures up the idea of a pleurisy or pneumonia, of a meningitis or encephalitis; nay, even of a conjunctivitis or iritis, with all the attendant symptoms, subjective as well as objective, functional disturbance and terminations, I admit that the objection is well founded. But if, on the contrary, all that we venture to deduce from our microscopical observations on the web of the frog in the inflamed state, be merely something concerning the general nature of the inflammatory process, something of the state of the blood and the blood-vessels in the inflamed part; something, in

short, in elucidation of the question as to the *proximate cause* of inflammation, so much agitated by the pathologists of the last century, then I apprehend that the objection is ill founded.

Still, there can be no doubt, that even as regards the simple elucidation of the state of the blood and the blood-vessels in an inflamed part of the human body, microscopical observations made on a transparent part of the warm-blooded mammal would be more satisfactory than similar observations on the cold-blooded reptile, especially when we take into consideration the marked peculiarity of character which the red blood-corpuscles of the mammifera present.

Accordingly, in pursuing my researches into the nature of the inflammatory process, I have not failed to direct attention to the microscopical study of the effects of wounds on the state of the blood and the blood-vessels in the web of the bat's wing, the bat being the only mammiferous animal which presents an external part of the body thin and transparent enough for microscopical examination.

Some results of this study, and also some results of an experimental inquiry into the influence of the nervous system on the state of the blood-vessels in inflammation, I beg leave to lay before the Royal Medical and Chirurgical Society, confident that any observations, however imperfect, relating to so important a subject in medical and surgical science, as inflammation, can never be without interest to them.

Part I.

The interdigital webs of the bat's wing are composed of duplicatures of skin with cellular substance, blood-vessels, nerves, and bands of elastic tissue interposed.

On either side of the elongated metacarpal bones and digital phalanges, there run an artery and corresponding vein, the trunks respectively of the arteries and veins of the webs.[1]

[1] For a detailed account of the distribution and arrangement of the blood-vessels in the webs of the bat's wing, see a paper in the 'Philo-

The arteries and veins closely accompany each other, a nerve only intervening between them.

In regard to the connection between the ultimate arterial ramifications and the capillary network on the one hand, and between the capillary network and venous radicles on the other; in the frog, I have elsewhere[1] observed, that, although the capillaries all communicate together, a given part of the capillary network receives its blood more especially and directly from the branches of a particular artery, and pours its blood more especially and directly into particular venous radicles. This arrangement exists also in the bat's wing, though not so unconditionally; for in consequence of the more free anastomosis of the arteries with each other, and of the veins with each other, the part of the capillary network which receives blood from the branches of a particular artery, and transmits it to particular venous radicles, is more circumscribed.

The veins of the bat's wing, which are furnished with valves, are remarkable for their rythmical or heart-like contractions. The contractions of the arteries are tonic. At the same time that the arteries and veins thus differ in respect to their mode of contracting, their muscular coat, which is the seat of the contractility, differs in respect to the microscopical characters of its component fibres.[2]

As to the capillaries, I have not observed any change take place in the width of their bore attributable to contractility of their wall, which, as in the frog, appears to possess but a single coat.

The blood-corpuscles of the bat, both red and colourless,

sophical Transactions' for 1852, entitled:—"Discovery that the veins of the bat's wing (which are furnished with valves) are endowed with rythmical contractility, and that the onward flow of blood is accelerated by each contraction."—A copy of which I beg herewith to present to the Society.

[1] On the State of the Blood and Blood-vessels in Inflammation, ascertained by experiments, injections, and observations by the Microscope, in 'Guy's Hospital Reports,' second series, vol. vii, part I, p. 6.

[2] See the paper above referred to, in the 'Philosophical Transactions' for 1852.

resemble those of the blood of man in shape and structure. The red corpuscles are about $\frac{1}{4200}$th of an inch in diameter. They are seen under the microscope to agglomerate into rolls, like those of human blood, not only in blood out of the body, but also in blood within the vessels, under the conditions to be mentioned below. The colourless corpuscles occur in the forms of both granule and nucleated cells. Measured while stagnant within the vessels of the living animal, the colourless corpuscles were full $\frac{1}{3600}$th of an inch in diameter. In blood out of the body, the granule cells when distended with water, measured about $\frac{1}{3000}$th of an inch; the nucleated cells about $\frac{1}{3200}$th of an inch, their nuclei being about $\frac{1}{4800}$th of an inch.

In the perfectly undisturbed state, the flow of blood in the arteries of the bat's web is uniform and rapid; so rapid that individual corpuscles cannot be recognised. In consequence of the free anastomosis of the arteries with each other, we may see the blood, in a given artery, flowing sometimes in one direction, sometimes in another. Often, we see the blood flowing backwards for a considerable distance in one artery, and then, at last, turning into an anastomosing artery, proceed in a direct course.

The arteries being capable, by virtue of the tonic contractility of their walls, of varying in width, the rapidity of the flow of blood in them is subject to variation, independently of any variation in the force of the heart's action. When the constriction of an artery is excited by slight pressure over it, if the vessel has not closed in, the flow of blood, though retarded, may continue direct; but it is sometimes seen to become retrograde, and, after continuing so for a more or less considerable distance, then joins the direct stream in an anastomosing artery.

If the constriction be so great that the artery becomes nearly or wholly closed in, the flow of blood ceases altogether at the place; but into the artery below the constriction, a stream enters in a retrograde direction by one branch and escapes in a direct course by another. From the artery above the constriction, the stream passes off by the first con-

siderable branch. The disturbance of the flow of blood now described as a consequence of constriction of an artery to closure, is similar to that which we shall find occasioned by section of an artery. In both cases, also, the resulting impediment to the flow of blood in the capillaries is similar.

When an artery is much dilated, a condition which may be induced by the application to the surface of the web of some irritant, such as a solution of sulphate of copper with vinum opii, the stream of blood is seen to be more or less accelerated. This important fact was discovered independently by Mr. Paget in the bat,[1] and by myself in the frog.[2]

In consequence of the rythmical contractions of the veins, and the pressure of valves, the flow of blood in these vessels is remittent, an acceleration taking place at each contraction. When the force of the heart's action does not bear very energetically on the veins, in consequence of the circulation in the arteries and capillaries not being quite free, the flow of blood in the first-named vessels becomes intermittent, being now chiefly maintained by their own rythmical contractions.[3]

The capillary circulation is seen to be accelerated or retarded, according to the degree of freedom with which the blood flows in the arteries and veins. The capillaries themselves are mere passive tubes.

As to the manner in which the blood-corpuscles comport themselves within the vessels:

Within arteries and veins, the red corpuscles keep together in the axis of the stream, and do not manifest any tendency to adhere to the walls of the vessels like the colourless corpuscles. When there is an impediment to the

[1] Lectures on Inflammation, at the Royal College of Surgeons, in 1850.

[2] The Essay in 'Guy's Hospital Reports,' ut supra. This essay, it may be mentioned, left my hands before Christmas, 1849.

[3] For a detailed account of the flow of blood in the veins of the bat's wing, see the paper in the 'Philosophical Transactions,' already referred to.

onward flow of blood, the red corpuscles agglomerate together in rolls similar to what we see in blood removed from the body; but when the blood is again permitted to flow freely, the rolls are broken up and the corpuscles are carried along confusedly mixed together. In the stream within the capillaries, the red corpuscles may often be seen arranged in a linear series, overlapping each other's edges; but on the occurrence of any impediment to the flow of blood, the red corpuscles become aggregated, so that we may sometimes see a single long roll occupying the axis of a capillary.

Colourless corpuscles may be seen adhering to, or rolling or sliding sluggishly along the walls of the vessels. I have observed this both in arteries and in veins. Towards the end of a protracted sitting, after the web had been much irritated, I have seen, in the venous radicles especially, colourless corpuscles accumulated in great numbers, as we so often see them in the frog. This, however, I have not found to occur so frequently in the bat.

Part II.

Having premised these observations on the distribution, structure, and endowments of the blood-vessels of the bat's web, and on the phenomena of the circulation of the blood in them, we are prepared to enter on the consideration of the effects of wounds of the web on the state of the blood and the blood-vessels implicated.

An artery, on being cut across, becomes constricted upwards in the direction of its trunk, and downwards in the direction of its ultimate ramifications; and so far as this constriction extends, the flow of blood in the vessel is arrested. In a minute or two, however, relaxation of the wall of the artery, and dilatation of its bore, are observed to take place, and then we find that in the upper part of the artery, the flow of blood becomes re-established as far down as the first considerable branch proceeding from it above the place of section: by this branch the stream passes off.

Below the wound, a retrograde stream of blood is kept up in the cut artery through the medium of an anastomosing vessel; but this retrograde stream passes off in a direct course by branches leading from the artery. The branches of the artery by which the blood enters in a retrograde direction, and those by which it passes out in a direct course, vary according to the mode in which the particular vessel happens to ramify at the place of section.

The retrograde stream flows more or less sluggishly, and the blood is loaded with red corpuscles.

I have not found veins become constricted after section like arteries.

In the upper segment of a divided vein, there is no further flow of blood up to the first considerable branch which joins the vessel above the wound. From the vein below the place of section, the blood which enters in a natural direction by one set of radicles flows out in a retrograde course by another set.

The re-establishment of the flow of blood in the upper segment of the artery, it has been said, extends only as far down as the first considerable branch immediately above the wound, although the artery may have become dilated down to that place. If there has been no bleeding from the cut vessel, the part of it between the wound and the branch above by which the stream passes off, is frequently seen to contain, at first, few or no corpuscles; but eventually, a greater or less number of corpuscles accumulate within the vessel, principally on one side, where they may be observed oscillating up and down.

In the artery below the section, the retrograde stream may not reach up as far as the wound, though up to that place the artery may have become dilated. An accumulation of blood-corpuscles, however, takes place similar to that just described in the vessel immediately above the wound.

These accumulations of blood within the cut ends of the artery form internal clots.

From the stream of blood which enters the upper segment of a divided vein, as above described, corpuscles enter the

part of the vessel next the wound, where they may be observed oscillating.

The lower part of the divided vein may not be at first filled with blood-corpuscles as far up as the wound, but in general, an accumulation eventually takes place, as in the artery below the wound.

The effect on the circulation in the part to which the divided vessels lead, is:

The blood in the last arterial ramifications, in the capillaries, and in the venous radicles flows tardily, and becomes loaded with red corpuscles which aggregate together. In some of the vessels, there takes place actual stagnation of the aggregated corpuscles.

This effect is explained by the circuitous route the blood has to follow through anastomosing branches to arrive at the last arterial ramifications, and to escape from the venous radicles. The blood moving slowly in consequence of diminished *vis a tergo*, the red corpuscles are allowed to aggregate, and being so aggregated, they move on less readily than the fluid plasma. The result is that more and more red corpuscles accumulate, until, in the vessels least under the influence of *vis a tergo*, the accumulation is so great that stagnation takes place.

In consequence of the free anastomosis of the vessels, the adjacent parts participate more or less in the obstruction of the circulation, more so perhaps than is the case in the frog, though, on the other hand, the obstruction in the part implicated, is, for the same reason, less in the bat than in the frog.

The effects of wounds of the web of the bat's wing on the state of the blood and the blood-vessels implicated, which I have now traced, are, in all essential respects, similar to those which I described in 1850, as occurring in the web of the frog's foot.[1]

The effect of constriction of the small arteries in inducing congestion of blood-corpuscles in the capillaries and venous

[1] Guy's Hospital Reports, ut supra.

radicles, is the same also in the bat as in the frog. I may add, I have observed a similar effect in the mesentery of the mouse.

The application therefore to the human body, which I formerly ventured to make of my observations on the frog, finds support in every respect, from the observations on the bat now recorded.

Part III.

Though I think there can be no doubt that the inflammatory redness of, let us say, the human conjunctiva, excited by a wound, by cold, or by the irritation of a foreign particle in the eye, is owing to congestion of red corpuscles in the vessels, and that the blood, thus loaded with red corpuscles, flows sluggishly in some of the vessels, and is in others altogether stagnant; and though I think there can be no doubt that this state of matters is induced in the same manner as that in which I have described it to be brought about in the frog and bat, still, it may be a question whether a sluggish flow or stagnation of blood in the small vessels be really the condition on which the redness, reputed inflammatory, always depends in the human body, or whether there are not cases in which the redness essentially depends on a dilated state of the arteries, and a more copious and freer flow of blood in the part?

In reference to this question, I offer the following observations:

In my Essay in 'Guy's Hospital Reports,' already referred to, an effect of section of the ischiatic nerve in the frog is stated to be dilatation of the arteries and a fuller and more rapid circulation of the blood in the web. To give an example:—The ischiatic nerve of the left leg of a frog being divided, the arteries of the web were found, on examination, dilated, and the stream of blood in them fuller and more rapid. The blood in the capillaries and veins especially appeared to be unusually loaded with red corpuscles. The relaxed arteries yielding more readily to the blood injected into them at each stroke of the heart, their pulsations were

more evident. The general effect to the naked eye, was increased redness of the web. Four days after the arteries were found still dilated, and the circulation in the web very free; epidermis exfoliated.

On the right or uninjured side, the arteries and circulation in the web remained unaffected, and the epidermis did not exfoliate.

Besides exfoliation of the epidermis, œdema of the leg and foot not unusually takes place after division of the ischiatic nerve; a tendency to ulceration is also manifested. But, on the other hand, I have observed that the granulations of a wound of the web are more luxuriant.

Here, it is to be observed, that the relaxation of the muscular coat of the arteries, on which their dilatation depends, appears to be the effect of section of the sympathetic fibrils bound up in the ischiatic nerve, and not of section of the proper spinal fibrils of that nerve. Thus, I find that if we lay open the lower part of the vertebral canal of a frog, and remove the roots of the nerves supplying the posterior extremities, together with the corresponding portion of the spinal marrow, so that all sensation, motion, and reflex action in the posterior extremities are abolished; the arteries are nevertheless seen, on examination of the webs under the microscope, to retain their contractility, nay, they appear even more disposed to become constricted. If now the ischiatic nerve (which comprises not only the spinal fibrils,—sensitive and motor,—the roots of which were with the corresponding portion of the spinal marrow destroyed, but also the fibrils from the sympathetic as yet uninjured), be divided on one side as high up in the thigh as possible, the result is that the skin of the extremity subjected to the experiment becomes, even to the naked eye, redder from vascular injection than that of the opposite extremity, and on examination of the web under the microscope, the arteries are found considerably dilated. In the web of the opposite extremity, on the contrary, the arteries are seen still much constricted—some even to closure.

Though after section of the ischiatic nerve, the arteries,

in these experiments, were not wholly deprived of their contractility, their tendency to become constricted was very small in comparison with that manifested by the arteries of the opposite extremity in which the ischiatic nerve was not cut.

The increased redness and heat of the ear and side of the head which supervene in Bernard's experiment of cutting the sympathetic in the neck—of a cat for example—can only be explained, I think, by supposing a fuller flow of blood in the parts in consequence of the arteries having become dilated from impaired contractility of their walls.

From the preceding facts and considerations, we are led to the conclusion, that the state described as inflammation of the eye supervening on section of the fifth pair, and of the lungs and stomach supervening on section of the par vagum, may possibly be owing to dilatation of the arteries (from paralysis of their muscular coat induced by section of the sympathetic fibrils contained in the nerves above named), and consequent fuller flow of blood.[1]

If this be so, we should have two different forms of disease reputed inflammation—the one primarily depending on a state of the blood-vessels and circulation of the affected part, and directly the converse of that on which the other depends.

However this may be, there is, I think, little doubt that the ordinary form of inflammation is that which depends, for its commencement, at least, on constriction of the small arteries, congestion of blood-corpuscles in the capillaries, and impeded circulation in the part.[2] In such a case, I have shown by experiment on the frog, that resolution is effected by the application to the web of a drop of vinum opii, or of any of the other agents which possess the property of causing dilatation of the arteries and acceleration of the

[1] In cases of disease implicating the fifth pair, for example, the sympathetic fibrils contained in it might not be destroyed, but merely irritated. In such case, constriction of the arteries of the eye, &c., it is to be inferred would be induced.

[2] See Guy's Hospital Reports, ut supra, p. 55.

flow of blood, an experiment which I adduced[1] as an interesting illustration of the *modus operandi* of stimulating collyria, applied to the eye for the cure of catarrhal ophthalmia.[2]

Considering that section of the ischiatic nerve has, for its effect, dilatation of the arteries and acceleration of the flow of blood, I subjected to this operation a frog in which I had previously excited inflammation of the web by the application of a solution of salt, in order to observe whether re-establishment of the circulation would be thereby promoted. The result was, that the circulation speedily became re-established. In another frog in which the second web of one of the feet presented considerable congestion and stagnation of blood-corpuscles, independently of any application of solution of salt, and in which a drop of vinum opii failed to produce resolution, the ischiatic nerve was cut as high up in the thigh as possible, whereupon the stagnation began to be dispersed.[3]

[1] Guy's Hospital Reports, pp. 49-50, 60.

[2] See also a paper, on the Nature and Treatment of Pustular Ophthalmia, as an illustration of Inflammation and the Healing process in their simplest manifestations, in the 'Medical Times and Gazette' for January, 1852.

[3] The activity of the flow of blood in the web after section of the ischiatic nerve, was not so great in the cases in which the roots of the spinal nerves with the corresponding portion of the spinal marrow were previously cut out, as in the cases here spoken of. The cause of this I have not yet investigated.

ON

INTERMITTING DIABETES,

AND ON THE

DIABETES OF OLD AGE.

BY

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It will be my object in the present paper, to point out some phenomena connected with Diabetes, which I do not find to be dwelt on by other writers; and as my facts bear on the theory of the disease, and indirectly on its treatment, they may be worthy of the attention of the Medical and Chirurgical Society.

Since the first part of this paper relates to the amount of sugar in the urine, I may be permitted to say a few words on the methods of determining the quantity that is present.

In the present state of our knowledge, no table whatever can be trusted; that is, it must never be considered sufficient to take the specific gravity, and thence to calculate the amount of sugar in the urine. It is no doubt true that this method may be employed in a single case, for the purpose of comparison, and that thus a distant approximation to the truth may be obtained; but for the comparison of different cases, this method is altogether worthless.

If diabetic urines were solutions of nothing but sugar in distilled water, such tables would give all the information wanted. But diabetic urines contain a multitude of other substances besides sugar, each of which is variable, and each

of which may cause the specific gravity to vary, whilst the quantity of sugar remains constant.

Hence, to be accurate, the amount of sugar must always be determined by direct experiment, and never calculated from the specific gravity. The following numbers will show to what an extent, in different diabetic urines of the same specific gravity, the quantity of sugar may vary; and also what different specific gravities may be found with a constant quantity of sugar.

In five cases I found the following numbers with Soleil's saccharometer.

	Urine, Specific Gravity.		Degrees of the Scale.	
1st Case.	1034·4	gave	6 =	about 6 grs. sugar in 1oz. urine
2d „	1034·8	„	16 = „ 16	„
3d „	1035·0	„	24 = „ 24	„
4th „	1034·0	„	16 = „ 16	„
5th „	1033·4	„	23 = „ 23	„

Again,

1st Case.	1042·0	„	30 = „ 30	„
2d „	1042·0	„	20 = „ 20	„
3d „	1043·4	„	13 = „ 13	„
4th „	1043·4	„	37 = „ 37	„

If examples are taken in which the quantity of sugar is nearly constant, the following variations in the specific gravity were observed:

	Urine, Specific Gravity.		Degrees of the Scale.	
1st Case.	1029·6	gave	5 = „ 5	„
2d „	1034·4	„	6 = „ 6	„
3d „	1033·0	„	7 = „ 7	„
4th „	1014·0	„	5 = „ 5	„
5th „	1027·8	„	5 = „ 5	„

Or again,

1st Case.	1031·0	contained	19 = „ 19	„
2d „	1034·8	„	16 = „ 16	„
3d „	1030·2	„	$17\frac{1}{2}$ = „ $17\frac{1}{2}$	„
4th „	1035·0	„	17 = „ 17	„

Or again,

	Urine, Specific Gravity.		Degrees of Scale.			
1st Case.	1036·8	contained	24 =	about	24	grs. sugar to 1 oz. urine
2d ,,	1044·4	,,	24 =	,,	24	,,
3d ,,	1033·4	,,	23 =	,,	23	,,
4th ,,	1035·0	,,	24 =	,,	24	,,

With still more sugar,

	Urine, Specific Gravity.		Degrees of Scale.			
1st Case.	1042·0	contained	30 =	,,	30	,,
2d ,,	1040·0	,,	30 =	,,	30	,,
3d ,,	1035·4	,,	30 =	,,	30	,,
4th ,,	1041·2	,,	29 =	,,	29	,,

Perhaps I shall show the false conclusions which would be drawn from calculating the quantity of sugar from the specific gravity, by the following numbers from two cases of diabetes. The analyses in the 1st case were made by Mr. Kemp, of Edinburgh, and the method of analysis was totally different from that which I used in the 2d case.

	Urine, Specific Gravity.		Grains of Sugar to the ounce of Urine.
1st Case.	1023	contained	2·2
	1025	,,	2·1
	1026	,,	4
	1027	,,	2·3
	1027	,,	4⅓
	1027	,,	4½
	1028	,,	14⅓
	1029	,,	3·3
	1029	,,	2½
	1029	,,	3
	1030	,,	3·6
	1030	,,	7¼
	1031	,,	14⅓
	1032	,,	17·5
	1033	,,	6
	1034	,,	8
	1035	,,	10
	1036	,,	5½
	1038	,,	7⅓

	Urine, Specific Gravity.		Degrees of Scale.		
2d Case.	1013·8	gave	5 = about	5 grs.	sugar to 1oz. urine.
	1014·4	,,	5 = ,,	5	,,
	1023·2	,,	14 = ,,	14	,,
	1025·6	,,	2 = ,,	2	,,
	1028·5	,,	4 = ,,	4	,,
	1029·6	,,	4 = ,,	4	,,
	1030·0	,,	15 = ,,	15	,,
	1031·0	,,	10 = ,,	10	,,
	1031·2	,,	2 = ,,	2	,,
	1032·2	,,	24 = ,,	24	,,
	1032·4	,,	16 = ,,	16	,,
	1033·4	,,	13 = ,,	13	,,
	1034·0	,,	16 = ,,	16	,,
	1035·4	,,	7 = ,,	7	,,
	1035·4	,,	17 = ,,	17	,,
	1037·8	,,	19 = ,,	19	,,
	1037·8	,,	20 = ,,	20	,,
	1038·3	,,	3 = ,,	3	,,
	1039·0	,,	20 = ,,	20	,,
	1043·4	,,	13 = ,,	13	,,
	1044·4	,,	24 = ,,	24	,,
	1044·8	,,	25 = ,,	25	,,
	1045·0	,,	13 = ,,	13	,,
	1045·4	,,	13 = ,,	13	,,
	1045·8	,,	25 = ,,	25	,,
	1048·6	,,	18 = ,,	18	,,

I have stated the degrees of the scale, as well as the calculated amounts of sugar, because some question as to the value of each degree may arise. The diabetic sugar on which M. Clerget experimented certainly appeared to me to be unusually pure.

Part I.

On Intermitting Diabetes.

There can be little doubt that our knowledge of the nature of diabetes may be extended, by means of accurate determinations of the variations in the amount of sugar in the urine passed at different periods of the day, and under different circumstances.

It is not my intention at present to enter at any length into

this question, though in one case the results of such experiments will be given. My object now is to record some cases in which, whether from the medical treatment, or the regimen, or from the natural course of the disease, the variation of the amount of sugar is not from much to little, but from highly saccharine to total absence of sugar.

The state of the urine a few hours after the sugar has disappeared, and an hour or two before it reappears, is most especially worthy of attention, inasmuch as it may lead to a truer knowledge of the state of the system which precedes the commencement of the diabetes. In intermitting diabetes, the disease may be seen beginning and ending; and the explanation of the state of the urine which precedes the appearance of the sugar and follows its disappearance, must be included in the true theory of diabetes. Moreover, a better knowledge of the antecedent phenomena may enable us to ward off the disease, if not to treat it with more success.

Case I.—A gentleman, 62 years of age, was sent to me by a physician, with a statement that sugar was present in the urine. February 18th, 1851, I examined the urine and found no trace of sugar by Trommer's test, nor by Liquor Potassæ test. I sent back the bottles to the physician, who in return said the secretion was in its reactions different from what he had tested on the 13th, when sugar was present.

We met in consultation March 22d, and on examination there was a reduction of the oxide of copper after long-continued boiling. The urine having just been passed, and being loaded with urates and containing an excess of urea, I considered that the reduction was probably caused by an excess of urates. He had lost two stone in three months; he had had considerable thirst, and a great appetite. Alkalies were recommended.

He wrote to me again in November, saying that his medical man in the North of England found no sugar, and that he was quite well.

Feb. 18th, 1852.—He came to me again saying that sugar had again been found; on examination none was present.

On the 29th of June I received by rail the urine passed on the 28th, specific gravity 1038, and it decidedly contained sugar. From the 18th of February he said he had remained very well, until within the last month, when "I found my old complaint returning. I have considerable thirst, and my mouth is occasionally parched, with a frequent desire to pass water; the quantity discharged is about two imperial quarts daily. I have lost no flesh and my appetite is almost too good. I feel very active on my limbs for the age of 63. I have a slight occasional pain above the right hip, but it is scarcely worth mentioning. My bowels are in a healthy state, and I go to the closet once daily."

He came to London on the 18th of August, and I found a small quantity of sugar.

Sept. 24th.—He wrote, I am very well, and my water stood not much above 1020 yesterday. My surgeon tested it before leaving home last week, and pronounced it free from sugar.

I heard nothing more of my patient until February, 1853, when his diabetic symptoms returned. The urine sent to me for examination on the 7th of February had a specific gravity of 1033·4, and gave 33 degrees of the scale = about 33 grs. of sugar in each ounce of urine. It was quite clear.

May 18th.—The urine collected in 24 hours, had specific gravity 1024; and with uric acid and oxalate of lime crystals, and some other crystals of unknown composition, contained an excess of urea, and gave no trace of sugar. The urine for several weeks has varied from 1016 to 1028.

Case II.—A very fat farmer, æt. 45, weighing 19 st. 2 lbs., who always ate excessively, had increased thirst for two months. He came to me May 29th, 1852. The urine passed the previous night was clear, specific gravity 1030. It gave a slight but decided trace of sugar.

The following morning, urine specific gravity 1035. Less sugar, but decidedly saccharine; gave a deposit of urates. Passes in 24 hours from 3 to 5 pints of urine. Tongue dryish, furred. Thinks he has more weakness of the muscles

than he has had. Sweats often. Bowels regular. Five years since had most violent attack of erysipelas. He was advised to take Carb. of Ammoniæ, gr. xv; Carb. Potassæ, gr. xxx; quotidie: and animal diet.

June 28th.—Was weighed on the 26th, and was 19 st. 3 lbs. Tongue less furred, from 3 to 3½ pints of urine. The quantity never exceeded 4 pints since May 29th. It was decidedly saccharine.

Sept. 15th.—The urine contained no sugar, specific gravity 1026. Contained oxalate of lime, and excess of urea.

April 20th, 1853.—I again saw the urine; it had specific gravity 1026, and contained no trace of sugar. He said that since his last visit the specific gravity had never been above 1030. He had had some red gravel, but for the last few days the urine had been quite clear. His weight was 19 st. 1 lb. He had had no thirst, the bowels were regular, and he felt very well.

Case III.—A clergyman, æt. 62, came to me January 20th, 1852, complaining of rather an increased flow of urine, which stained his clothes, and was sticky. He had lost flesh, and had drunk rather more than usual. He had no cough, and perspired very readily. He was red faced, and rather of a full habit. The urine had specific gravity 1036, and contained a little sugar. I restricted him to animal diet, and gave—Carb. Ammonia, gr. x; Carb. Soda, gr. x, ter die. Brandy and water only, to drink.

He came to me April 12th. Urine, specific gravity 1026·15, contains scarcely, if any, trace of sugar. I omitted the medicine.

May 18th.—Urine, specific gravity 1025, contains no trace of sugar. Has taken no medicine; has occasionally eaten a little more bread. Feels in every respect well.

Case IV.—On the 12th of April I was first asked to see, with Dr. Quain, a gentleman, æt. 33, who had come from India. He gave me the following account:—"In November, 1852, I first observed a great increase in the urine. One day I observed

the utensil in my dressing-room covered with red ants; the daily repetition of this in my house, and in my office, convinced me that the urine attracted the ants. I looked for sugar, and found it by Trommer's test. I represented this to my medical man, and at his request the urine was analysed, and no sugar was found in the urine passed that day, which happened to be mail day, always one of great excitement, and on it I had observed that but little urine was passed. Two days afterwards a considerable quantity of sugar was found. The quantity was about 120 ounces in twenty-four hours. The specific gravity was occasionally 1038, once 1040, usually 1034; with Dover's powders, and afterwards 24 grs. of Carb. of Ammonia daily, the quantity was reduced to 70 or 80 ounces, and at one time the sugar vanished, or nearly so. At the end of January the stomach was disordered, with some feverishness, and it was determined that I should come to England. I sailed the 27th of February; the quantity of urine was about 80 ounces, and sugar was present. I improved on the voyage; but at Gibralter I ate some oranges, which brought on a return of the symptoms. In not longer than twenty-four hours I passed 92 ounces of urine. The quantity generally was from 70 to 75 ounces until I arrived, April 6th."

April 9th.—I found the urine had a specific gravity 1038·4, and gave 27 degrees of scale = about 27 grains of sugar, to the ounce of urine. It contained oxalate of lime, urates, and an excess of urea. He was advised—

℞ Ammonia Bicarb., ʒiv;
Potassæ Bicarb., ʒiij;
Tr. Card. C., ʒj;
Aquæ, ʒiij, cochl. j magnum ex aquâ ter die;

Sodæ Phosph., ʒss, primo mane;

Pil. Hydr., gr. iij, hâc et crastenâ nocte.

On the 19th the urine was scanty, loaded with urea, and contained scarcely a trace, if any, of sugar.

℞ Potassæ Bicarb., ʒiiss;
Ammoniæ Sesqui-Carb., ʒj;
Tr. Zingib., ʒj;
Aquæ, ʒiij, cochl. j amplum ex aqua bis die.

On the 29th the urine had specific gravity 1034·4, and gave 6 degrees of scale = about 6 grains of sugar to the ounce of urine. It was loaded with urates.

On the 28th the urine had specific gravity 1031·4, and contained no trace of sugar.

On the 29th:—

P.M.	Ounces.	Specific Gravity.			
2	10	1025·8	no sugar	much oxalate of lime	excess of urea.
4½	5	1027·4	„	much uric acid	„
8½	8	1029·3	„	„	„
10	4	1027·0	„	much oxalate	„

30th:—

5.45	13	1027·2	„	„	„
12	9	1025·8	„	much urate	„

On the 29th the breakfast consisted of fish, one egg, milk, brown bread. Lunch—Beef, lobster, small piece of brown bread, claret one glass (a small meal). Dinner—Fish, mutton, greens, cheese, claret (ate sparingly).

30th.—Breakfast. Eggs, herring, milk, bread.

May 1st:—

		Ounces.	Sp. Grav.	
Urine passed	12 noon	8	1025	a doubtful trace of sugar, excess of urea.
„	2.30 p.m.	7	1026	no trace of sugar, excess of urea.
„	4.30 „	6	1025	„ „
„	8.30 „	9	1030	„ acid.
„	10.30 „	10	1026	„ nearly neutral.
„	3 a.m.	11	1023	„ deposit of phos. lime.

2d:—

Urine passed	7 „	13	1025	„	highly acid.

Breakfast—Ham, milk, brown toast. Lunch—Cold beef. Dinner—Fish, soup, hare, spinach, a little claret, bread and butter, tea, little milk.

May 8th.—Urine passed this day, on which more bread was eaten, specific gravity 1033·4, gave 4 degrees of scale = about 4 grains of sugar to an ounce of urine.

May 18th :—

		Ounces.	Sp. Gravity.		
Urine passed at	7 a.m.	10	1029	no sugar.	
„	9 „	4	1032	„	
	and at				
„	5 p.m.	11	1030	„	excess of urea.
„	12 „	3	1035	„	„

19th :[1]—

„	8 a.m.	15	1031	„	„

Case v.—A lady, æt. 50 (?), weighing about 14 stone, had always suffered from indigestion. She had felt at Christmas weaker than usual, and at the end of January she caught a severe cold. By this she was much exhausted. She had an aphthous state of the tonsils, and a great dryness of the tongue. "It was like a piece of wood." At the same time excessive thirst came on. The bowels were excessively confined. She took quinine and acids. At the beginning of April the quantity of water attracted notice. It was probably at least ten pints in the twenty-four hours.

I first saw this lady on the 11th of April; she had much thirst, and great debility. The urine was highly acid, specific gravity 1042, and gave 30 degrees of scale = about 30 grains of sugar to the ounce of urine. It contained an excess of urea, and gave a deposit of uric acid crystals.

She was put on a strict animal diet, and was given Carb. Ammoniæ, Carb. Potassæ, ter die.

On the 18th the thirst was considerably less. The quantity of urine was less. It was not examined.

On the 25th the urine made the night previous had specific gravity 1029·6, and gave 5 degrees of scale = about 5 grains of sugar to the ounce.

The urine made the following morning had a specific gravity 1031·0, and gave 19 degrees of scale = about 19 grains of sugar to the ounce. This was accounted for probably by a small quantity of bread which was taken during the night. The thirst was very much less.

[1] On two occasions, in June and July, I again found sugar in the urine. He left London August 4th. In a letter dated October 8th he states that, by the most careful examination of the urine, he finds that sugar is absent except when he takes bread for breakfast.

On the 26th	there was still a slight trace of sugar,	sp. gr.	1030
„ 27th	there was no trace of sugar	„	1025
„ 28th	a trace of sugar	„	1026
„ 29th,	still trace	„	1028
„ 30th,	morning, a trace of sugar	„	1030
„ 30th,	night, no sugar	„	1010
May 4th,	no sugar	„	1020
„ 9th	„	„	1025
„ 10th	„	„	1024
„ 11th	„	„	1025
„ 13th	„	„	1028·7
„ 14th,	morning, no sugar	„	1026
„ 14th,	night „	„	1025
„ 15th	„	„	1026
„ 19th[1]	„	„	1028

Case vi.—A very extensive farmer, æt. 37, came to me October 27th, 1852. Six years previously he had great irritation of the urinary organs, having to make water thirty times an hour. He never passed any blood. He first found an increased flow of urine from the 10th to the 20th of last August. The quantity was two gallons of urine daily. He had excessive dryness of the throat; and in five days, he lost 14lbs. The weight of the urine had been 1050. When he drank water the mouth was perfectly dry, as if he had taken nothing. This state lasted two weeks. Has been subject to violent bilious headaches.

The urine now is very thick from urates. Specific gravity, 1038. Contains decidedly some sugar, and an

[1]

May 26th,	no sugar		sp. gr.	1026·6
„ 27th,	„		„	1033·4
„ 28th,	„		„	1026·0
June 9th,	„	red sand	„	1028·6
„ 10th,	„	„	„	1028·8
„ 11th,	morning „	„	„	1024·0
„ 11th,	night „	„	„	1016·0
July 7th,	night „	„	„	1026·0
„ 8th,	„	„	„	1022·0
Oct. 7th,	„	„	„	—

excess of urea, so that it crystallises out immediately with nitric acid. No albumen. No pus. Complains of very much flatulence. The bowels act regularly daily. Weight now 12 st. 1 or 2 lb.

Has been a great eater of common fruit puddings. Vegetables in great quantity. Always greens and much potatoes, with very little meat, twice daily, but little each time. For the last six years he has drunk no beer or port wine; they caused irritable symptoms. Takes brandy and water. Can walk three or four miles. Red faced and ruddy, but fallen away. For two or three years sexual desire has gradually decreased. Has taken excessive exercise.

December 25th.—He adhered most rigidly to dietetic directions, and to the treatment until the end of November, when he was passing only about 14 ounces a day. Specific gravity, 1040. His appetite failed him, and he was losing flesh and strength, and getting depressed in spirits, besides which, he was suffering very severely and constantly from excessive and most distressing irritability of the bladder. The urine being very acid, and densely loaded with lithates and excess of urea, he was given more fluid.

℞ Potass. Bicarb., ʒj;
Tr. Hyoscy., ʒj;
Inf. Buchu, ʒiv. M. Sumat ʒj ter die.

This was taken from November 30, with great relief to the irritable state and diabetes; he now passed 25 to 30 ounces, specific gravity varying from 1010 to 1020, and without sugar. The daily averages from the 18th were 1028, 27, 23, 21, 29, 30, 29.

The prostate was found enlarged on examination, and very painful to the touch.

The urine sent to me, December 25th, contained no sugar. Two specimens were examined.

January 31st.—Four specimens of urine passed this day.

		Specific Gravity.	Degrees of Scale.			
5½ ounces	at 3 p.m.	1031·4	gave 5 =	about 5	grs. of sugar	per ounce.
6½ „	7 „	1035·4	„ 7 =	„ 7	„	„
10 „	9 „	1032·4	„ 16 =	„ 16	„	„
7 „	6 a.m.	1038·3	„ 3 =	„ 3	„	„

"He has been going on much the same since last report. Sometimes passing water with little or no trace of sugar; and on one or two occasions, with evidence of a large quantity. The last few days he is weaker and much more depressed. Appetite good. Taking Inf. Buchu, Tr. Hyoscy., and Acid. Nitro-Muriat., for large quantity of water (sic)."

March 27th.—The report was not so favorable. He said he had adhered most rigidly to the diet, not having eaten eight ounces of vegetables since October. His stomach, he said, detested animal food. Cream, he said, increased the thirst, and made the mouth foul. The usual quantity of liquid taken in twenty-four hours would not amount to one pint.

26th.—The water passed was sent to me.

		Urine.	Specific Gravity.	Degrees of Scale.	Sugar to the ounce.
5 p.m.	he took two eggs and a mutton chop.				
7 „	. . . . .	7 oz.	1045·4	gave 13 =	about 13 grs.
10 „	mutton chop and lettuces.				
10.15 „	. . . . .	7 „	1045·8	„ 25 =	„ 25 „

27th:—

3.30 a.m.	. . . . .	9 „	1044·4	„ 24 =	„ 24 „
6 „	wing of a guinea fowl.				
6.45 „	. . . . .	3½ „	1045·4	„ 13 =	„ 13 „
9 „	2 eggs and a chop.				
10.30 „	. . . . .	9 „	1043·4	„ 24 =	„ 24 „
1 p.m.	boiled rabbit.				
1.15 „	. . . . .	6½ „	1044·8	„ 25 =	„ 25 „
3 „	. . . . .	2½ „	1048·6	„ 18 =	„ 18 „

In each of these specimens nitric acid caused an immediate crystallisation of nitrate of urea, and in all there was free uric acid and oxalate of lime.

May 13th.—For two days, nothing except milk was taken, no other food whatever. Last food was eaten at 9 p.m., May 12th.

Urine passed before

	Milk.	Urine.	Specific Gravity.	Degrees of Scale.	Sugar to the ounce.
6½ a.m.	22 oz.	. . .	1030·8 gave	1 =	about 1 grs.
8 „ bowels acted		4 oz.	1035·4 „	17 =	„ 17 „
9¼ „ . . .	22 „				
9¾ „ bowels acted		4 „	1037·8 „	20 =	„ 20 „
1¼ p.m. . .		7 „	1037·8 „	19 =	„ 19 „
1½ „ . . .	22 „				
3 „ . . .	11 „				
6 p.m. . . .	11 „	10½ oz.	1034·0 „	16 =	„ 16 „
7½ „ . . .	11 „				
9 „ . . .	22 „				
9½ „ went to bed		6 „	1039·0 „	20 =	„ 20 „
12¾ „ . . .	11 „	15½ „	1014·4 „	5 =	„ 5 „
4¼ „ . . .	6 „	14 „	1013·8 „	5 =	„ 5 „
	138 „	61 „			

Every specimen, except the last two, contained an excess of urea, and free uric acid and oxalate of lime.

14th :—

	Milk.	Urine.	Specific Gravity.	Degrees of Scale.	Sugar to the ounce.	Contained excess of urea, uric acid, and oxalate of lime.
7 a.m.	11 oz.					
7 „		8½ oz.	1023·2 gave	14 =	about 14 grs.	„
9¼ „	11 „					
1 p.m.	11 „	8½ „	1032 „	24 =	„ 24 „	„
5 „	22 „					
5¾ „		5 „	1030 „	15 =	„ 15 „	„
9¼ „	22 „					
9¾ „		4½ „	1028·5 „	4 =	„ 4 „	„
4½ a.m.	11 „	8 „	1025·6 „	2 =	„ 2 „	„
	88 „	34½ „				

So that at the end of two complete days, in which milk and nothing else was taken, only two grains of sugar existed in each ounce of urine.

15th.—For two days the diet consisted of fish only. Codfish either boiled or fried, eaten without bread, or vegetable, or sauce of any kind whatever.

		Urine.	Specific Gravity.	Degrees of Scale.	Sugar to the ounce.
7 a.m.	fish . . .	4 oz.	1031·0	gave 10 =	about 10 grs.
8 „	fluid mag., 1½ oz.; 3 oz. Bristol water.				
9 „	bowels acted slightly, fish .	5½ „	lost.		
12¼ „	5 oz. Bristol water.				
1¼ p.m.	fish.				
2¼ „	11 oz. Bristol water.				
3 p.m.	bowels acted slightly . .	10½ oz.	1031·2	„ 2 = „	2 „
3¾ „	11 oz. soda water.				
5½ „	fish.				
5¾ „	. . . . .	7½ „	1029·2	„ ½ = „	½ „
6¾ „	11 oz. Bristol water.				
9 „	fish.				
9¼ „	. . . . .	8 „	1029·6	„ no sugar	

16th :—

3¼ a.m.	. . . . .	14 „	1028·9	„	„
6¾ „	fish.				
7 „	bowels acted slightly . .	4½ „	1030·1	„	„
8 „	11 oz. Bristol water.				
9 „	. . . . .	5½ „	1026·4	„	„
9½ „	fish.				
10¾ „	6 oz. Bristol water				
11 „	. . . . .	4 „	1026·0	„	„
2 p.m.	. . . . .	6½ „	1029·1	„	„
2¼ „	fish.				
3¼ „	11 oz. Bristol water.				
4¾ „	11 oz. soda water.				
5¾ „	fish . . .	7½ „	1030	„	„
9 p.m.	. . . . .	8½ „	1023·0	„	„

17th :—

		Urine.	Specific Gravity.	Sugar.	Excess of urea, uric acid, oxalates.
1¼ a.m.	. . . . .	6½ oz.	1030·0	none	„

For two days butcher's meat and nothing else was taken.

6 a.m.	mutton chop.				
6¼ „	. . . . .	5½ „	1028·0	„	„
7½ „	11 oz. Bristol water				
9 „					
10 „	. . . . .	5½ „	1027·0	„	„

		Urine.	Specific Gravity.	Sugar.	Excess of urea, uric acid, oxalates.
12½ a.m.	11 oz. Bristol water	4 oz.	1030·0	none	,,
1½ p.m.	tripe.				
3¾ ,,	5 oz. Bristol water .	6 ,,	1030	,,	,,
5½ ,,	mutton.				
9 ,,	lamb.				
9½ ,,	11 oz. Bristol water	10 ,,	1031	,,	,,
18th :—					
1½ a.m.	. . . .	9 ,,	1027	,,	,,
7 ,,	mutton chop.				
7¼ ,,	. . . .	8 ,,	1029	,,	,,
8 ,,	11 oz. Bristol water.				
9 ,,	mutton chop.				
11 ,,	. . . .	5½ ,,	1028	,,	,,
1½ p.m	mutton chop.				
2½ ,,	. . . .	6½ ,,	1029	,,	,,
2¾ ,,	11 oz. soda water.				
5½ ,,	mutton.				
6 ,,	. . . .	5½ ,,	1030	,,	,,
7 ,,	11 oz. Bristol water.				
9 ,,	mutton.				
9½ ,,	. . . .	6½ ,,	1029	,,	,,
3¼ a.m.[1]	. . .	10 ,,	1028	,,	,,

Case vii.—A fat woman, who lived well, in the neighbourhood of Oxford, and was a patient of Dr. Jackson's for diabetes in the autumn of 1847.

"Nov. 15th.—Urine, specific gravity of 1043.

,, 22d. ,, ,, 1030.

,, 24th. ,, ,, 1026.

,, 26th.—The patient is much better in health and spirits. Diet, meat, eggs, port wine, greens, and a few mouthfuls of bread. The urine has changed so much in the last fortnight as to astonish me; what I send, when I first saw it appeared to have a quantity of grease floating on the surface, a thickish cloud at the bottom, and the middle clear."

On examination, the sediment, which was very great, consisted of multitudes of uric acid crystals, oxalate of lime octohedra, and epithelium. The specific gravity was 1025·2,

[1] See Postscript.

and with sulphate of copper and liquor potassæ, it gave marked evidence of sugar.

Grains.		Grains. Per 1000
51,260 boiled with Ch. of Barium & Nit. Acid;	Sul. of Baryta	=3·82=7·45
51,260 precip. by Ch. of Calium & Amm.;	total Phos. of Lime	=2·02=3·94

Nov. 16th, 1848.—I received another specimen of this urine, containing oxalate of lime and sugar. Dr. Jackson says, "She was a patient of mine this time last year, and then had all the symptoms of diabetes, and the urine contained sugar. Under treatment the health improved and the sugar disappeared. She was under treatment about four months, and seemed to have got quite well, and continued so all through the summer. About a fortnight ago she sent for me, with a return of all her symptoms, though nothing like the same extent as last year.

"The first specimen I examined contained a little sugar, specific gravity 1041, and the quantity was about six pints in twenty-four hours. The second was specific gravity 1036, and about five pints. The third I sent you (this had specific gravity 1041·3), it was passed just before going to bed. In those twenty-four hours she must have passed about four and a half pints. Her health has improved in the fortnight: last year I should think she lost two stone in weight, which to all appearance she has quite regained."

The disappearance of the sugar from the urine in the cases which I have related, is in itself a matter of some interest; but the state of the urine voided after the sugar has disappeared, is still more worthy of attention.

The remarkable excess of urea which was so frequently found before and after the sugar disappeared, might perhaps be attributed to the continued animal diet, but the occurrence of free uric acid, and the oxalate of lime in the urine, point most clearly to a state of indigestion which is so often to be found, without any sugar appearing in the urine.

The state of the urine appears to me to mark the passage of diabetic indigestion into acid indigestion, as distinctly as the change is sometimes stated by the patient himself. "I used to have excessive disorder of the stomach; I could

hardly eat anything in consequence of the acidity and uneasiness which I suffered, but now since the water has increased, the disorder of the stomach has disappeared. I can eat anything. I never suffer now from acidity, but I am losing flesh and strength.

The following theoretical contrast between ordinary acid and saccharine indigestion may suggest some questions for solution.

Ordinary indigestion shows itself in a want of action on the sugar and starch taken as food, in consequence of which excessive acidity is produced; that is, the changes in the non-nitrogenous food are imperfect. Imperfect changes also occur in the nitrogenous food; this is made evident by an excess of urates and urea in the urine, and perhaps also by the formation of oxalate of lime.

In diabetic indigestion the effect may be also traced on the two great classes of food. At first, from the non-nitrogenous food, sugar is formed instead of acids. Ultimately, if not simultaneously, sometimes the arrest of healthy changes extends to the albuminous food, and instead of an excess of urates and urea, other products are formed, one of which is sugar. It is possible that some of the other products may still be found in the urine.

Whether this theory be true or not, it is practically useful to remark the tendency to acidity and excess of urea in these cases of intermitting diabetes. In such cases, animal diet alone or with alkalies, may stop the formation of sugar. It follows also that when oxalate of lime, uric acid, and excess of urea are found in the urine, it is probable that the diabetes may be temporarily, if not permanently, removed.

The presence or absence of these substances in the urine may lead to the recognition of the stage of the disease, and they may thus guide us in prognosis and in treatment.

Part II.

I pass on now to the frequency of diabetes in old age.

In a paper on the habitual presence of sugar in the urine of old people, read before the Academie de Médecine

de Belgique, by M. Dechambre, it is stated that experiments were made by him on 20 aged women in the Salpetrière, and that in 19 there was decidedly sugar in the urine.

The sulphate of copper test was chiefly used. Occasionally the fermentation test was employed.

M. Dechambre concludes, that sugar is habitually present in the urine of old people. Now though I cannot find any proof of this in the experiments which I have made on the urine of some aged persons, yet on referring to the notes which I had taken of some of my diabetic patients during the last three years, I was surprised to find in how many of the cases life was far advanced. It appeared to me still more worthy of note, that the symptoms were in almost all of these cases modified in intensity.

Among the notes of 29 cases of diabetes, I found 11 were above 60 years of age, and 6 of these were above 70 years old. Of these 11 cases—

In 2 the disease was intermitting; they are cases I and III in Part I.
„ 6 the quantity of urine was scarcely if at all increased in quantity.
„ 1 the quantity was increased, but the disease had probably existed for 16 years.
„ 1 albuminous urine was present, and the diabetic symptoms were very slight.
„ 1 above 74 years of age the disease existed in its intensity.

11 cases, in 10 of which the disease was so slight, that the general symptoms hardly declared it.

Case I.—A gentleman, æt. 70, first consulted Dr. Prout in 1828, and again two years ago he went to him for diabetes. Has a very ruddy healthy look, stout, and has not lost weight; is now 13 stone. Complains of frequency and quantity in making water: 15th and 17th of January passed 4 pints and upwards of urine, drank 3 pints. Has been thirsty all his life. Tongue reddish. Pulse 84. Bowels regular. Always perspired a great deal. Does not know what bilious attack, stomach attack, or headache is.

Dr. Prout allowed him to drink Bass's ale.

The least particle of camphor causes irritation and redness of the skin, and the effect will not go off for several weeks.

The external use of a liniment of camphor, soap, and opadeldoc have caused it.

He was put on Carb. of Ammonia and compound soap and Opium pill. Strict diet.

April 8th.—Does not pass so much urine: considerably less. On the 6th and 7th he drank two pints and a half, and passed three pints and two pints and a half of urine. The urgency does not come on so violently. Appetite still very good, but the troublesome and intense thirst has gone off. Has not taken the pill. Very little sugar in the urine.

April 28th.—Took two grains of Pil. Saponis c. Opio, which caused violent irritation of the skin for twenty-four hours. The intense thirst is now gone.

August 13th, 1845, he weighed 12 st. 11 lbs.; October 10th, 1845, 12 st. 5 lbs.; August 2d, 1847, 13 st. 3 lbs.; February 5th, 1851, 12 st. 3½ lbs.; June 4th, 1852, 11 st. 12 lbs.

May 4th, 1852.—He made the following observations on the quantity of water drunk and the quantity of urine passed.

	Water.	Urine.
January 15th, in 24 hours	Drank 2 wine bottles.	Passed 2¼ bottles.
„ 17th, in 24 „	„ 2 „	Nearly 3 „
April 6—7th, in 40 „	„ 3 „	„ 4 „
May 2d—3d, in 48 „	„ 4 „	„ 4 „

Complains of pain after a short walk in the muscles of the legs.

August 9th, 1852.—Two quantities of urine examined, specific gravity 1025 to 1030.

November 22d.—Urine specific gravity 1022, contains a trace of sugar. Went to Vichy, drank there three half-pint glasses of Grand Grille before breakfast, three glasses of Celestin afterwards. Bath for three quarters of an hour. Took Bordeaux wine with water. Still complaining of pain in the legs. Weighs 13 lbs. less than when he went away. The bowels were not confined by the Vichy water.

February 4th, 1853.—He came to me complaining, that yesterday the left foot became cold and numbed, and that he had excessive pain down the leg, so severe that he hardly

slept at all. He says the foot feels as if there was no life in it, and it is certainly paler and less sensitive than the other.

February 9th.—Says he found relief from the pain by getting out of bed. The quantity of urine is two pints and three quarters daily, and this is the exact quantity which he drinks.

February 15th.—Still very severe pain. The urine has specific gravity 1027·8, and gave 5 degrees of scale = about 5 grains of sugar to the ounce of urine. It gave a plentiful deposit of urates. The pain increased: the little and next toe on the left foot mortified. He lost his appetite. The urine became scanty, excessively loaded, and deep coloured, and I found no sugar in it ten days previous to his death, March 25th.

The disappearance of the sugar from the urine when the appetite failed, and the disease was hastening to its end, is very remarkable; but I have in many cases found, that when any feverish attack comes on, and the appetite is much diminished, the quantity of sugar in the urine is usually very much reduced, so that when the physician would say the patient was worse, the chemist would say he was decidedly better. As the fever subsides, and the appetite returns, the sugar increases, though no more vegetable food may be taken.[1]

Case II.—A physician, æt. 76, told me that he had consulted Dr. Prout, and had certainly had sugar in the urine for five years, and probably much longer. About four years since he confined himself to an exclusively animal diet, and in four or five months he lost 40lbs., but he considers that he has nearly regained his loss since. He now lives chiefly on animal food, taking a biscuit in two days. The

[1] A short and very remarkable case is mentioned in the 'Comtes Rendus,' Dec. 28th, 1848. A patient of M. Rayers, in the Charité,

Dec. 18th, had sugar . . 18·5 degrees of scale.
„ 20th, attacked with pleuro-pneumony.
„ 22d, had sugar . . 6 „
„ 27th, inflammation much increased; no sugar.
„ 28th, he died.

quantity of urine on the 1st of January, 1853, was about the average. The specific gravity was 1040. There was sugar decidedly.

I prescribed Carb. of Ammonia alone, in 10 grain doses thrice daily.

Case III.—A gentleman, æt. 77, "tolerably active yet," has been affected with diabetes for six or seven years, though only in a moderate degree. The disease is still moderate, though rather on the increase. Usual specific gravity of the urine 1035. The urine contains "more uric acid than sugar, although twelve drops of liquor potassæ are taken every other day." The tongue is generally more or less white, and he has rather a tendency to costiveness. The liver being a little torpid, he takes vegetable aperients, and $2\frac{1}{2}$ grains of blue pill occasionally.

The urine contained sugar, and uric acid crystals.

Case IV.—A gentleman, æt. 77, consulted me for diabetes. Thinks he has "had sweet water" for two months. All last summer he had thirst. For the last twelve or fourteen years he had occasional, but slight attacks of gout. His son has had the gout severely since he was fifteen; he is now nearly fifty. Complains now of thirst; urine saccharine, specific gravity 1033, contains also uric acid crystals; does not pass in 24 hours above a pint and three quarters. His face is florid. The pulse 80, soft. The tongue clean. He has fallen away, and has had a carbuncle lately on the face. Can eat anything, nothing ever disagrees with him.

In a few weeks he became drowsy, comatose, and died.

Case V.—A gentleman, æt. 63, December 14th, 1847, under the care of Dr. Watson, who sent me the following note:

"He calls himself well, and *seems* eminently so. Stout fat, and ruddy. Eats and drinks heartily and indiscriminately, and has no dyspepsia. Perspires copiously. Makes much urine, and often by day and by night, *and has done so these forty years*. Says his pulse occasionally intermits, and has long done so. I found it steady and 80 in the

minute. His father died with diseased heart at 77. My patient had some bronchial trouble last winter. Had lately some redness of the toe for a day or two. Comes to me that I may see what he is when well, as he means to put himself under my charge whenever he is ill. He had noticed some floating substance (not sand) in his urine, and was curious to know what it might be." I examined the urine and found it light coloured, and it contained torulæ, specific gravity = 1032·0. It gave slight but decided evidence of sugar with the copper test.

February 10th, 1848.—Urine, specific gravity 1030·2, contained sugar.

Grains.		per 1000.
51·510	boiled with Ch. of Barium & Nit. Acid; Sul. of Baryta=	2·17=4·21
51·510	precip. by Ch. of Calcium & Ammonia; Tot. Phosphates=	0·47=0·91

18th.—Received yesterday, bottles marked 1, 2, 3, and 4. No luncheon was taken.

			Specific Gravity.	
1,	made just before breakfast,	much mucus,	1027·6,	sugar.
2,	„ three hours after,	little mucus,	1024·4,	less sugar.
3,	„ just before dinner,	mucus,	1027·8,	sugar.
4,	„ three hours after,	„	1027·7,	„

May 10th, 1849.—More sugar in the urine.[1]

Case vi.—February 1850, I saw a gentleman 66 years of age who had passed many years in the East Indies. For six or seven years he had had more thirst than other people, and had passed an excess of water; the thirst had been more decided since Christmas twelvemonth; he had been wasting two years, and had lumbago with some fever since Christmas. For thirty years he had a remarkably good appetite. The urine was clear and pale, specific gravity 1027·4; it contained sugar and a trace of albumen.

Pulv. Doveri, grs. v, horâ somni omni nocte;
℞ Liq. Ammoniæ, ℥. xl;
Mist. Camph., ʒxij;
Coch. iij magna, bis die.

---

[1] July 28th, 1853, urine passed during the night . Specific Gravity. 1020·4 Degrees of Scale. gave 3 Sugar to the ounce. = 3 grs.
„ 29th, on getting up . 1021·0 „ 5 = 5 „

On the 13th of March the urine was specific gravity 1031·8. Still contained sugar. No albumen.

On the 14th of March, before food, specific gravity 1018·2. Still sugar. Doubtful if any albumen.

He states that he has much less thirst and no more frequency than when in perfect health. In point of quantity he passes much less. Thinks that he is much stronger, and feels more healthy altogether. He takes no sugar, no vegetables, and a little bread, milk, butter, eggs, fish, and meat. Has continued the medicine. Weight now 14 stone; has weighed 16 stone 8 lb. A little perspiration yesterday.

July 10th.—The urine has specific gravity 1023·9, contains the slightest trace of sugar. The quantity of urine now is not more than one half what it was. Could not be absent two hours from home before, now never feels any urgency. Passes the night from eleven to four without being disturbed. Has continued to gain decidedly. Was near writing to ask whether he was not making too little water. Weighed a fortnight ago 14 stone 2 lb. Thirst gone, does not drink one-third what he used to drink.

Has continued the

Dover's powder, 4 grs.;
Also Liq. Ammoniæ, ʒj;
Mist. Camph., ʒxij;
Coch. iij, bis die.

June 11th, 1852.—Has remained well, the urine is specific gravity 1024·5; contains the slightest trace of sugar. Has felt well, but was given the sulphuret of arsenic, which he took for a fortnight. At the end of this time he felt "something unusual," and he gave it up. The quantity of water was reduced by it; now the quantity is about 40 ounces. He drinks but little. He weighed 14 stone. Does not think that he has lost flesh lately. Has continued the ammonia off and on for two years.

Case vii.—A gentleman, æt. 73, who had lived very freely in his youth, had been a patient of Dr. Prout's for diabetes for five years.

He had very little thirst. Passed in twenty-four hours six pints of fluid. He complained of much tenderness in the loins. He considered that he had been diabetic for sixteen years. All vegetable food was stopped, and the sugar entirely disappeared.

April 25th.—Specific gravity 1035. Has taken 10 grains of Carb. Ammoniæ after each meal. Thinks he is much better for it.

Sept. 30th, 1852.—Still says that he thinks all his symptoms have been the same for sixteen years. He has not lost flesh. The urine still contains sugar. He eats a good deal of bread, and drinks a pint of wine at least daily. He complains of pains in the thighs, worse he says after walking. In twenty-four hours he passed eight pints of urine, specific gravity, 1030.

Case viii.—A gentleman, æt. 62. For a year and a half had suffered from carbuncles, and during that time he had been falling away. He has had frequency in passing the water, rising four times at night. The bowels were confined, and he complained much of giddiness and pain in the head, for which he had a seton in his neck. The urine had specific gravity 1034. Triple phosphate crystals, albumen, blood-globules, and sugar, in small but distinct quantity, were present. Torulæ were evident in twenty hours.

April 29th, 1848.—Some œdema of the legs.

November 22d.—Urine more albuminous, specific gravity 1029. Still contains sugar.

December 14th.—Specific gravity 1028·9, albuminous and saccharine.

April 11th, 1849.—Urine, specific gravity 1016·6, contains albumen. With copper test, or with liquor potassa test, shows scarcely a trace of sugar. Gave a deposit of urates on standing.

April 16th.—Urine, specific gravity 1021, no precipitate of urates; gives evidence of more sugar; albumen as before.

May 1st.—Passes a pint and a half of urine in twenty-four hours. Specific gravity 1022·4. With the microscope multi-

tudes of uric acid crystals were seen, with very coarse fibrinous casts. There was much albumen and very little sugar.

The symptoms of Bright's disease increased, with cough and expectoration, and he died comatose on May 18th.

Examined 17 hours after death.—The body was much emaciated. The parietes of the chest and abdomen were thickly coated with yellow fat. There was no fluid in the abdominal cavity. The kidneys were very adherent to the fatty substance surrounding them. The surface of the right kidney was smooth, and mottled with deep depressions. Some cysts and some stellated veins were very marked. On section, the small arteries of the cortical structure were open, and the substance was not wasted. The left kidney contained more cysts and depressions of a darker colour, with blood streaks extending from them. No fibrinous deposits were visible in cutting through the depressions. The kidneys were markedly mottled.

The pancreas contained much interstitial yellow fat, and did not look healthy. The liver was healthy, with good looking bile. The stomach was large, and very thin at the larger curvature, but not pulpy; it did not break on pressure. The blood in the veins was dark; not so elsewhere.

There was an immense cavity as big as the fist in the apex of the right lung, and tubercles in all the upper lobe. The left lung was full of tubercles, with some small vomicæ.

The heart was full size, with dilated cavities; not much, if at all enlarged. The aortic valves were slightly thickened, and the auricles contained an immense clot.

Case ix.—A patient, æt. 74, said that thirteen weeks before his admission into St. George's Hospital, he was well. He had been working in ditches a few days, when he became very hungry and thirsty all day long, with pain in the loins. During the last week, the usual quantity of urine was ten pints in twenty-four hours. At present it is fourteen pints. He is much emaciated. Pulse regular, languid, 74. He tasted his urine, and said it was sweet as honey. During the

first week, the quantity of urine was twelve pints in twenty-four hours. At the end of the week after his admission he was seized with pain in the head and feverish symptoms. The urine decreased to five pints. At the end of another week the feverish symptoms were removed, and the urine again increased to thirteen pints in twenty-four hours. It continued about this quantity until the end of the third week, when he became dull and heavy and very tremulous, when the urine diminished to two pints. The quantity never increased; his tongue became dry, he remained in a state of muttering delirium, becoming at last comatose, ten days after the urine so suddenly decreased in quantity. There was considerable effusion of serum under the arachnoid, which was opaque, and there was much fluid in the lateral ventricles of the brain.

The pericardium and pleura on the right side were inflamed and coated with lymph. Both cavities contained a large quantity of yellow serum, with flakes of lymph. The kidneys were healthy.

I have mentioned the last case only to show, that in old age the disease sometimes exists in great intensity; still the other cases warrant the conclusion, that diabetes most frequently occurs in old age without the very marked symptoms which generally lead to the recognition of the complaint. It is very probable that this modified diabetes may sometimes be the cause of that debility which is attributed to old age alone; and perhaps by chemical examination of the urine in advanced life, this disease may be detected far more frequently than at any other period, and then possibly by an animal diet, the strength may be preserved, and the life prolonged.

The occurrence of diabetes in old age points also to the theory of diabetes, as an indigestion resulting from an arrest of healthy changes in the food. This, at present, imperfect and very general formula of the disease requires to be worked out by careful experiments. The most suitable cases for investigation, are those in which the disease is modified by old age, or so imperfectly established, that a

more healthy state of urine may be seen from time to time to occur.

In those more confirmed cases in which the sugar is most probably formed from the nitrogenous, as well as the non-nitrogenous constituents of our food, investigation may, perhaps with advantage, be directed to the discovery of the substances which are complementary to the sugar, and the variations in the amount of urea in these cases may give important results.

M. Bernard has based a theory of diabetes on his most remarkable experiments. According to him, an altered condition of the action of the nerves on the liver causes an excessive formation of sugar in that organ; according to another theory, in consequence of the altered action of the nerves, the sugar of the food is not changed in the liver into fat. These statements, though more precise, are not to me so probable as the theory that diabetes is an indigestion, or, in the language of Dr. Prout, an error in the converting power of the stomach. There can be no doubt that M. Bernard's experiments will be extended, and then a more exact theory of the disease may include an affection of the stomach as well as the liver.

As regards the treatment in almost all the cases mentioned in this paper, alkalies were used with benefit. Whatever is beneficial for excessive acidity is still more useful in diabetes. Small meals; free from sugar and acid, and the substances which can give rise to sugar and acids, constitute the best diet.

I have more than once been told by patients that potash could not be taken, and that soda and ammonia agreed perfectly. Vegetable acids with alkalies are unobjectionable.

I may here state the conclusion from some experiments which are yet unpublished, that porter contains from 20 to 40 grains of sugar in each ounce of liquid; ale from 12 to 130 grains; port wine contains much sugar; sherry, less sugar; claret, none. Also that the absence of all sugar, and the presence of little alcohol, causes claret to taste highly acid, whilst the quantity of acid in good claret is not

much more, and sometimes is less, than in other kinds of wines which have no acid taste, for example, most port wine.

The mixture of claret and Vichy water allays the thirst in diabetes better than any liquid that I have prescribed.

Postscript; *October*, 1853.

Case vi.—I again requested this gentleman to try the effect of an animal and milk diet.

May 22d.—The second day of an exclusively animal diet.

	Food and drink.	Urine.	Sp. Gr.	Sugar.	Appearance.
6 a.m.	4 oz. Bristol water	11 oz.	1024·8	none	red sand and excess of urea.
7 „	mutton				
7½ „	. .	1 „	1024·6	„	„
9 „	mutton .				
11½ „	11 oz. Bristol water	6 „			
1 p.m.	chicken				
2½ „	. .	4½ „	1024·0	„	„
4 „	11 oz. Bristol water				
5 „	mutton				
7¾ „	. .	9 „	1026·9	„	„
8 „	11 oz. Bristol water				
10 „	. .	4 „	1026·2	„	„
	37 oz.	35½ oz.			

May 23d.—Exclusively milk diet.

5½ a.m.	. .	13 oz.	1024·6	„	„
6½ „	milk, 22 oz.				
10 „	„ 11 „				
10½ „	bowels moved	8½ „	1029·4	7 deg.=7 grs. to oz.	„
11 „	milk, 11 oz.				
1½ p.m.	„ 11 „				
3 „	fluid magnesia, 2 oz.				
3½ „	. .	7 „	1029·5	1 „ =1 gr.	„ „
5½ „	milk, 22 oz.				
9 „	„ 11 „				
10 „	. .	8½ „	1027·2	doubtful	„

May 24th:—

1½ a.m.	. .	8½ „	1010·0	very doubtful	no red sand nor excess of urea.
	88 oz. milk 2 „ magnesia	45½ oz.			

	Food and drink.	Urine.	Sp. Gr.	Sugar.	Appearance.
6 a.m.	bowels moved	5½ „	1018·8	no sugar	no red sand
6¼ „	22 oz. milk,				nor excess
	2 „ fluid magnesia				of urea.
7½ „	. .	8½ „	1009·2	2 deg.=2 grs. to oz.	„
10 „	11oz. milk; bowels moved				
12 „	. .	11½ „	1014·8	½ „ = ½ „	„
1 p.m	22 „				
3¼ „	. .	7½ „	1014·4	no sugar	„
6 „	22 „				
7½ „	. .	11 „	1010·8	„	„
9 „	22 „				
10 „	. .	7 „	1009·8	„	„
1¼ a.m.	. .	20 „	1004·8	„	„
	99 oz. milk	71 oz. urine			
	4 „ magnesia				

He said—"My kidneys and bladder are always most comfortable under milk diet, but I frequently have a little heartburn; but I do not feel the same morbid irritability of stomach that I do when living entirely on animal food." As I wished still further to try the effect of milk diet, he undertook to take nothing else for a week. The daily quantity was from 100 oz. to 130 oz., and on one day 149 oz. He passed from 50 oz. to 90 oz. of urine.

Urine passed at	Specific Gravity.	
June 17th, 7 a.m., before milk diet was begun,	1029	contained no sugar; red sand
„ 18th, 7 „ . . .	1016	contained no sugar
„ 19th, 7 „ . . .	102	11 deg.=1 gr. to oz.
„ 20th, 7 „ . . .	1020	under 1 deg.=1 „
„ 21st, 7 „ . . .	1016	trace
„ 21st, 12 midday . . .	1005	„
„ 22d, 7 a.m. . . .	1017·6	no trace
„ 23d, 7 „ . . .	1015·0	„
„ 24th, 7 „ . . .	1034·2	„

Animal diet only was taken on the 23d.

July 23d.—His pulse was 72; his tongue clean; he looked florid and well; had no irritation of the bladder; could take tolerable exercise; had gained 8 lb. in weight, being now 12 st. 10 lb. He said that, from the time of his first visit to me, all the flour he had eaten would not one quarter fill his hat, and he had not taken a pocketful of vegetables. The urine was perfectly free from sugar.

AN ACCOUNT

OF A

DISSECTION OF AN OVARIAN CYST

WHICH CONTAINED BRAIN.

BY

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Received June 20th.—Read June 28th, 1853.

THE attention of morbid anatomists and pathologists has been directed for many years to the structure of Ovarian Cysts and their contents, and especially to those which apparently contained fœtal remains, with a view of determining, if possible, their origin and mode of growth. At present, however, these researches have not been crowned with success, probably from the comparatively limited number of observations that have at present been recorded, the results of which still leave us in much doubt as to their mode of origin. It is from this circumstance that I have ventured to bring this case before the notice of the Society, in order that at some future time it may be of some service in attempting to explain the law which governs the singular development and mode of growth of these tumours, and also from the fact of its being the first time (as far as I have been able to ascertain), that such contents have been noticed, as contained in ovarian cysts.

The patient, who was the subject of this disease, was a female, single, æt. 28, who was admitted into St. George's

Hospital, with typhus fever, under Dr. Bence Jones, on May 14th, and died six days after her admission.

I have thought it perfectly unnecessary to record here the history of the case, as it was totally unconnected with the disease found in the ovary.

I have also been unable to find that she presented during life any symptoms, which led to the suspicion that such disease existed during life.

The post-mortem examination was made twenty-four hours after death.

General Appearances.—The body was well-formed and in good condition. The skin of both legs and arms was covered with numerous small petechial spots; and at the lower part of the abdomen, the skin presented numerous minute vesicles, containing a perfectly clear watery fluid.

Thorax.—There were no pleuritic adhesions. No fluid was found in either pleural cavity. The lungs were very much congested throughout. The heart and valves were healthy. The blood was quite fluid.

Abdomen.—The stomach was healthy. Numerous patches of ecchymosis were found in the mucous membrane of the lower part of the ileum, but no ulceration was observed. In the cæcum, two or three distinct ecchymosed patches were seen in the mucous membrane, and on one of these this membrane was very distinctly ulcerated, in the form of three exceedingly small linear patches. The liver was healthy. The spleen was congested. The kidneys were also congested, but otherwise healthy.

Pelvis.—The vagina was healthy. The uterus was somewhat larger than natural, and its muscular wall thickened. The membrane lining its cavity was covered throughout its entire extent with a highly vascular villous membrane, presenting a great resemblance to the decidua. This membrane lined its entire extent, being limited to the upper part of the neck of the uterus below, and to either uterine extremity of the fallopian tube above. The round ligaments were both healthy. The right fallopian tube was somewhat more dilated than natural, towards its distal extremity, and its

cavity contained a thin bloody fluid, it was pervious throughout. The left fallopian tube was healthy. The right ovary was healthy.

The left ovary could not be seen, its position being occupied by a large cyst, the size of an orange, which was situated at the upper and left side of the pelvic cavity. The upper half of this was transparent, and contained about two ounces of a light straw-coloured fluid. The lining membrane of the interior of the cyst was vascular. Its wall was composed of an external peritoneal layer, an inner smooth skin and shining coat devoid of epithelium, and an intervening fibrous layer capable of separation into several laminæ. On its inner wall were seen several small masses of fat, which were found lying between the lining membrane and the fibrous layer. The cavity of the cyst contained a few short light brown hairs. The lower half of the tumour consisted of a second cyst containing some yellowish white purulent looking fluid, and a considerable amount of granular fatty matter, of the consistence of honey, consisting of fat, with some scaly epithelium, and having intermixed with it numerous fine hairs of a light brownish colour. From some experiments performed by Dr. Noad and myself, it was ascertained that this fatty matter was fluid at less than the natural temperature of the body. Being placed in a freezing mixture, at the temperature of 40° F. it became of the consistence of honey. Upon being heated to 74°, it was of the consistence of gruel, and became perfectly liquid, but was still opaque at 80°. It boiled at 220°.

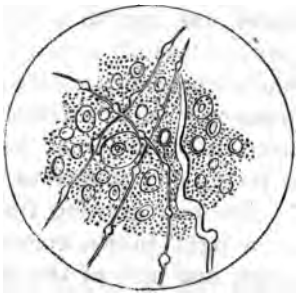
The hair upon being separated from the fatty matter was found to exist in considerable quantity, part being free and mixed with the fat, part being attached to the wall of the cyst, into which they were inserted by means of distinct bulbs. It was of a light brown colour, the individual hairs varying in length from a quarter of an inch to nineteen inches, and most of them presenting a blunt and also a sharp point. Some of the hair upon being fused with carbonate of soda, and tested with the nitro-prusside of sodium, showed distinct traces of sulphur.

At one part of the bottom of the latter cyst, a portion of the wall presented an appearance similar to the surface of the scalp. This portion was irregular in form, about two inches in length, but its breadth differed at various parts, its opaque appearance was due to a thick layer of scaly epithelium, mixed with fat which covered its surface. On removing this, the surface beneath presented a fine reticulated appearance, and numerous small circular spaces, through which the hairs protruded, the fibrous web composing this layer contained in its meshes numerous sebaceous glands and hair follicles. The hair growing from this portion of integument was of a light brown colour, and about five inches long. Beneath one part of the skin a quantity of fat was deposited in the areolar tissue beneath. A canine tooth projected from the skin fully formed, and the neck of which was surrounded by a thin, loose continuation of the skin; it was implanted in a fragment of bone covered with periosteum, which was reflected into the socket of the tooth. The fragment of bone was placed beneath that portion of the sac which corresponded to the skin; its upper surface, which projected towards the cyst, had a convex outline, was rough, irregular, and presented a distinct socket, in which the tooth above mentioned was implanted. Its lower surface was smooth and concave. Its structure appeared to resemble in every respect that of true bone.

Projecting into the floor of this sac, was observed a third cyst, it was about the size of a walnut, and situated near to the fragment of bone above mentioned; the wall of the cyst was delicate, and its inner surface highly vascular, the vessels were large, forming numerous meshes, which resembled somewhat those of the pia mater. The contents of this cyst consisted of a softish, white substance, the surface of which was perfectly smooth, and presented all the characters of brain. On microscopic examination it was found to consist entirely of the ordinary elements of nervous matter, being composed of a very large number of varicose nerve tubules of varying size, intermixed with the elementary

components of grey matter, viz., nuclei, and nucleated vesicles, containing granules.

Fig. 1.



Varicose nerve tubules, and ganglion globules, composing the mass of brain, found in the ovarian cyst.

On removing the contents of this cyst, a fourth was observed projecting into its floor; this contained a yellowish white opaque gelatinous mass, of the consistence of the white of egg, containing a large number of granular cells, and free granules. At the side of the latter and between it, and the wall of the parent cyst, a fifth cyst was observed, containing a semifluid light coloured mass, which also presented numerous nerve tubules and ganglion globules. The mass contained in this cyst was much less like brain matter than the contents of the cyst previously described, differing from it by being of a more fluid consistence, and by its colour, which was of a dirty greenish yellow tinge. The ordinary elements of brain were also less numerous than in the other cyst.

As far as I have been able to ascertain, nervous matter has never been previously found as forming a part of the contents of these cysts. It is from this circumstance that I have deemed it right to bring this case before the notice of the Society, as it may assist, to a certain extent at some future period, in explaining the law which governs the development of these remarkable tumours.

AN ACCOUNT OF AN INSTANCE OF REMARKABLE DEFORMITY OF THE LOWER LIMBS.

BY

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Received June 27th.—Read June 27th, 1853.

As the opportunities of examining malformations of the human body so as to make known the anatomical condition of the parts must necessarily be few, I am induced to consider the account of a single and rare instance of monstrosity worthy of presentation to this Society. In this case, whose peculiarities I will notice presently in detail, the deformity affected the lower limbs, but chiefly the thighs, and it involved the bones, the muscles, the joints, and the blood-vessels. Thus the thigh-bone was absent, except a rudiment of the lower end on the left side, and its corresponding joints were likewise wanting; the muscles were altered in various respects; the large femoral artery was transferred from the front to the back of the limb; and not the least interesting particulars in the history are the advanced age (55 years), and the extraordinary strength and activity of the person.

These peculiarities existed in a man named Harvey Leitch, or Leach; and our knowledge of them is due to the unusual circumstance of his body being given by himself to

the late Mr. Liston for examination.[1] He was well known on the stage in his day, and appeared in the guise of imps and monkeys. It may be within the recollection of some of the Fellows of this Society, that he was an object of interest and curiosity in this metropolis a few years before his death in 1847, when he feigned to be a newly-discovered animal, and invited inquiry into his nature by the question, "What is it?" in public hand-bills.

In the outward shape the body was natural in the upper, but deformed in the lower part. Thus the upper half, including the trunk, was well fashioned, and corresponded to the like part of an athletic man of five feet seven or eight inches, with rather long arms, for the measurement across the chest to the tips of the fingers gave five feet eleven inches. But the lower members were withered, though in an unequal degree, and the right was shorter and much more malformed than the left one. The disproportion between the upper and the lower halves of the man, and the aptitude of the bodily form for the creeping or climbing movements, will be apparent on reference to fig. 1, which has been most obligingly drawn by Mr. Ford from a cast in the Museum of University College. The following were the measurements of the body:—On the side of the longest limb (the left), the length was four feet, but of this the trunk forms the greatest part; for, from the top of the head to the front of the pubes, the measurement was two feet nine inches; whilst the right limb was about a foot, and the left was longer by only three or four inches, when it was in a half-flexed position. In consequence of the great difference in length between the upper and lower members, the hands could be brought to the ground almost during the

[1] At the time of the examination, notes of the anatomical condition were taken by myself, and some sketches of the parts were made by my late valued friend and colleague, John Phillips Potter, with the view to publication. And through the kindness of his father, the Rev. J. P. Potter, to whom I take this opportunity of expressing my obligations, I have since been furnished with those drawings, but their incompleteness has interfered with their use in illustrating this paper.

erect attitude, so as to assist in progression, and the body could be easily converted, by means of a suitable covering, into a form resembling that of a baboon. In spite of the imperfect state of the lower limbs, there was great personal activity; and as the body was supported almost altogether on the left limb, the right foot touching the ground only with the toes, most of the movement must have been derived from it and the arms.

Bones.—The deficiency in the bony framework is confined to the lower part of the body, and is greater on the right than the left side, in correspondence with the greater alteration in the external form.

The *pelvis* is rather below the average, both in size and strength. In each os innominatum the acetabulum is wanting, and the place of that articular cavity is occupied by a small hole or depression, rather less than half an inch in diameter. In each bone, also, the lower and anterior spine of the ilium is widened, and is covered by a smooth surface for articulation with one or two small irregular pieces of bone, *a* (rudiments of the trochanters), to which the flexor muscles were attached. These facts and others respecting the form and connections of the bones are well illustrated in the drawings (Plate II) by Mr. Ford, from the skeleton.

No *femur* exists in the right limb; and only a fragment of the lower end of that bone, measuring two inches in depth and three in width, is present in the left. The patella was not found on either side.

Tibia and fibula.—In the second segment of the limbs both the bones are developed, but they are more malformed on the right than the left side. The right tibia is widely forked above (Plate II, fig. 2), and each point is attached by muscle and ligament to the pelvis, but without any articulation or even contact; the outer point, which is the continuation of the shaft of the bone, is anchylosed with the head of the fibula.[1] The corresponding bones of the left leg are nearly

[1] A somewhat similarly-shaped femur is in the museum of University

of the usual size, but they are slightly deformed above, and are rather bent in their shafts.

In the right *foot* the natural arch is much increased, and the phalanges can be placed at right angles to the metatarsal bones, as they are represented in Plate II. In the left foot the articulation between the astragalus and the os calcis is surrounded by osteophytic growths, as if to give increased security to the joint.

Articulations.—In consequence of the absence of the cavity of the acetabulum and of the part of the femur that naturally fits into it, there was not any hip-joint on either side, so that the connection between the bones of the limbs and the pelvic arch was effected by muscle and ligament, but without contact.

In the right limb the two branches of the tibia were directed upwards towards the pelvis, one point being near the front of the pubes, the other behind the front of the innominate bone, and were fixed in those positions by fibrous bands and muscles, whilst at the midpoint of the fork the bone was further united to the pelvis by other ligamentous bands. In the left limb the remnant of the femur projected upwards amongst the muscles, and was connected by ligamentous structures to the front of the innominate bone: on this left side the knee-joint was nearly complete, only the patella being absent.

At the front of the os innominatum, between the smooth surface over the lower spine and the loose portions of bone before referred to (Plate II), was a large, loose, multilocular sac or articulation, over which the flexors and extensors of the limb were spread.

Muscles.—Alterations in the muscular system were proportioned to the malformation in the bones, and some muscles were so transformed or joined with others, that the task of distinguishing them was not always easy.

The flexors of the thigh had the usual arrangement at their origin, and were attached below on both sides, in

College, London, No. 2990; it was obtained from a deformed limb in the dissecting room.

the absence of the femur, to the loose piece of bone (analogue of the trochanter) that articulates with the front of the os innominatum. The direct extensor (gluteus maximus) takes its ordinary origin, but varies in its insertion on the two sides; for, on the right, where the thigh bone is altogether wanting, it is fixed into the upper and outer point of the tibia; and, on the left, into the fragmentary lower end of the femur.

The abductors and rotators of the thigh were inserted on both sides, into the loose piece of bone before mentioned. The adductors in the right limb, only two inches in length, were attached to the inner point of the tibia; whilst in the left, where they were rather longer, they were connected with the remnant of the femur.

The extensors of the knee-joint disappeared on the right side with the absence of the articulation and the femur, but were present on the left, though they were united to the end of the femur above the joint, and may thus give an explanation of the disappearance of the patella. The flexors of the same articulation were in usual force in the left, or more complete limb, but my notes do not state the condition in the right one.

Between the osseous and the muscular systems a mutual dependence is thus observable. For where the femur is absent, as on the one side, the muscles that are usually attached to it are either wanting, or, if present in a rudimentary condition, they are inserted into the contiguous bones. And where a fragment of the thigh-bone remains, as in the left limb, its muscles are developed in a corresponding degree.

Blood-vessels.—A great departure from the usual arrangement was found in the distribution of the blood-vessels; inasmuch as the leading anterior femoral vessel was absent from the usual place, and in its stead a large artery was developed at the back of the thigh with the sciatic nerve. Further, the known proportion between the internal and external iliac arteries was reversed, and the former became, contrary to the rule, the channel through which the blood was supplied to the greater part of the lower limb. The following was the disposition of the vessels:

The external iliac artery measured only one quarter of an inch in diameter, and furnishing, as is its wont, the epigastric and circumflex iliac offsets, left the abdomen beneath Poupart's ligament. In the thigh the vessel passed deeply amongst the adductor muscles, like the profunda artery, and distributed the circumflex and the other branches belonging commonly to the profunda; this unusual artery represented in part the femoral, by its continuity with the external iliac trunk, and, in part, the deep muscular artery of the thigh, by its position, and the manner of its branching.

The internal iliac artery was half an inch in diameter, and, after passing through the pelvis, where it supplied the usual visceral and parietal branches to that cavity, it issued below the pyriformis muscle with the great sciatic nerve. At the back of the thigh it accompanied that large nerve, and bifurcated below the knee-joint into the anterior and posterior tibial trunks. In the left, or more perfect limb, the muscular connections of the artery were like those of the companion nerve. This large posterior femoral vessel, by which the blood was conveyed through the first segment of the limb to the other segments, occupies for a given extent the position of the sciatic artery; but beyond the region of the buttock it has no analogy to any known vessel, for an artery distinct from the nerve is not recognised in the present state of our knowledge.

A very large internal saphenous vein was present, and this opened into a deeper trunk, lying by the side of the anterior artery of the thigh. My notes do not refer to the farther condition of the veins; but in a case which has been reported by M. Caillard,[1] the large venous trunk of the limb ascended from the popliteal vein, along the back of the limb with the unusual artery as far as the small trochanter: at this level it was directed forwards through the adductor muscle to the usual position of the femoral vein at the top of the thigh.[2]

The above-mentioned arrangement of the arteries of the

[1] A reference to this will be found farther on at p. 446.

[2] It is a question, whether this is the common arrangement of the

thigh, viz., the presence of two trunks—a small anterior and a large posterior—corresponds to that of the arteries of the lower limb of the bird, and presents us with a good illustration of the resemblance that the so-called varieties in man may bear to the condition of the parts in beings lower in the scale of the vertebrated class of animals. And as such deviations from the archetype do not occur without suggesting to us some hypothesis as to their mode of production, I am inclined to suppose that the present remarkable arterial arrangement may be due to the existence, in all cases, at an early period in the formation of the fœtus, of a temporary condition of the vessels resembling the permanent state in birds. Our facts seem to point to the probable development of a vessel at the back of the thigh, with the sciatic nerve, at some stage of fœtal life: for such a posterior vessel is constant in birds; it occurs also, rarely however, in man; and in the adult human body an artery always accompanies the nerve for a given distance. With the existence of the supposed vessel, for a certain period in the growth of the embryo, the observed peculiarities might be explained; since if there is the common distribution of the arteries of the limb, it may be supposed that the elongation of the posterior vessel is arrested, only the upper third remaining small and pervious, as the sciatic artery of the adult, whilst the femoral artery becomes of large size. But, if the unusual distribution is present, the reverse of this is the result, as in Harvey Leitch; for then it may be supposed that the posterior vessel (that with the nerve), is prolonged into the limb, and is increased in size, by its taking on the office of supplying blood to the leg and foot, in consequence of the anterior or femoral artery being interrupted in its growth by some disturbing influence.

Only three cases of a like departure from the typical form

vein with this unusual state of the artery. For the vein may have a like disposition without any peculiarity of the artery. (See Quain's 'Anatomy of the Arteries,' pl. viii, fig. 3.) In the Museum of the Royal College of Surgeons, Edinburgh, in the Barclay Collection, is also a preparation, No. 779, illustrating this exceptional distribution of the veins.

of the blood-vessels have I been able to find recorded. One is described by Dr. P. H. Green,[1] of which the preparation is said to be in the museum of La Pitié, Paris. Another has been observed by M. Caillard,[2] and has been published in his graduation thesis. And the third was noticed by M. Dubrueil,[3] Professor of Anatomy in Montpellier. In all three examples the peculiarity of the vessels existed only on one side of the body, two being on the right and one on the left; and it was present in well-formed bodies: in my case, on the contrary, the peculiarity was the same in both lower limbs, and occurred with deformity of the members. It appears, too, in accordance with the conclusions arrived at by Professor Quain,[4] that a similar deviation from the standard condition of the vessels is found much more commonly on one than on both sides of the body.

The surgical interest of this rare peculiarity of the blood-vessels has not been overlooked by those who have had the opportunity of observing it; but as the subject is not noticed in our anatomical and surgical works, I may perhaps be allowed to refer to the point, with the hope that my remarks may be the means of making it known to others. That such a state of the vessels might be a cause of much embarrassment to a surgeon in an amputation of the thigh, can be readily imagined; and that much blood might be lost, under some circumstances, before even the source of the hæmorrhage would be suspected by him who was unacquainted with this occasional peculiarity, is very probable. And in the case of an attempt to tie the femoral artery in the upper part of the thigh for an aneurism, or a wound lower in the limb, the operator might be much perplexed at not finding the large femoral artery, but only an inconsiderable artery after much

[1] In the Lancet of Feb. 18, 1832.

[2] Propositions de Médecine et de Chirurgie. Thèse, No. 307, p. 18; Paris, 1833.

[3] Des Anomalies Arterielles, with an Atlas of Plates, in 4to; Paris, 1847. See p. 36, and plates 11 and 12.

[4] The Anatomy of the Arteries, &c., with an Atlas of Plates, in folio; London, 1840-44.

search, the ligature of which would arrest neither the pulsation in the aneurism nor the bleeding from the wound.

Nerves.—The nerves of the limbs were regular in their number and position, and each of the three principal nerves (sciatic, anterior crural, and obturator,) was accompanied by an artery. My notes refer to the decrease in size of the obturator and the anterior crural.

My notice of this *monstrum informe* would be scarcely complete without a short reference to the place that it would occupy in a classification of so-called monsters. As the departure from the man-like form is limited to absence of parts of the lower members, this curious case would be included amongst those forms distinguished by deficiency of the limbs. Professor W. Vrolik has illustrated this kind of malformation in plates 76 and 77, of his work on Monstrosities.[1] In order to diminish the length of my paper in this particular, I may refer to the work above mentioned.

In conclusion I may remark, without entering into the consideration of the cause of the malformation, that the influence originating the unnatural state of the body in Harvey Leitch was confined almost altogether to the upper segment of the lower limbs, and was limited to the fore and central parts of that segment; for the posterior muscles of the thigh were less altered than the anterior, whilst a large vascular trunk was constructed at its back part as a substitute for an anterior, interrupted, and diminished vessel. The bodily imperfections seem, then, to be owing to a disturbance of the formative process during the sprouting of a certain part of each lower limb.

[1] Tabulæ ad illustrandam Embryogenesin Hominis et Mammalium.

EXPLANATION OF THE PLATES.

Plate I.

This drawing represents the outward shape of the man: it has been copied from a cast taken after death, but the artist has very skilfully raised the drooping head, and animated the face. During the taking of the cast, the right arm was supported by a string in its present position.

Plate II, Fig. 1.

In this sketch the lower deformed half of the skeleton, from the last dorsal vertebra downwards, is very well portrayed. The left limb has been drawn in an extended and straight position, instead of a bent and sloped one, as in the cast, so that it appears much longer in comparison with the other leg, than in fig. 1. The spine has been bent to the left side by the action of the muscles.

a. Separate pieces of bone, that articulate with a smooth surface on the lower and anterior spine of the os innominatum: two are on the left side, but only one on the right.

b. A small pit in the situation of the acetabulum, which was rather more than a quarter of an inch in diameter.

c. The remnant of the femur on the left side.

d. The left tibia; *d'*, the right one much altered.

Fig. 2.

A drawing of only the tibia and fibula of the right limb: the two are anchylosed at the upper articulation.

a. The fibula: it is separate from the other bone below, and enters largely into the ankle-joint.

b. The tibia, which is branched above into two points: by means of these points it was attached chiefly to the pelvis, but ligament was also inserted into the roughness at the centre of the fork.

OBSERVATIONS

ON

CYSTIC DISEASE OF THE TESTICLE.

BY

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Received June 27th.—Read June 28th, 1853.

THERE is no disease affecting the testicle which appears to be less understood than the Cystic, various opinions being entertained both of its nature and mode of origin. This obscurity has arisen chiefly from the late period of the disease at which the morbid specimens are examined; for, as they are obtained only after an operation which the surgeon delays until considerable disorganisation has taken place, the pathologist rarely possesses the opportunity of making an investigation in the early stage of the cystic development. Having recently removed a testicle, in which the disease was less advanced than usual, and having with the valuable aid of Mr. John Quekett succeeded in determining the mode in which the cysts are formed, I beg to communicate the results of these observations to the Society.

It is well known, that the cystic disease of the testicle was described by Sir A. Cooper, under the name of the "hydatid disease;" and yet this distinguished surgeon evidently supposed that the cysts might be formed of enlarged and obstructed tubuli, for he remarks, "although at first sight they appear to be cysts, yet when traced they are not distinct bags, but send out solid processes by which they are connected with other bags."[1] In this opinion I was disposed to concur, the disease appearing to me to be analogous

[1] Observations on the Diseases of the Testis, p. 83.

to the cystic tumours in the breast, which originate in a morbid dilatation of the lactiferous tubes.[1] But having subsequently observed in several specimens of cystic testicle, that the tubuli seminiferi in a healthy condition formed a layer spread over the morbid mass, I was at a loss to reconcile the tubular origin of the disease with this condition of the part until the difficulty was solved by the inquiries made in the following case.

M—, a tradesman from a town in Kent, æt. 37, of a pale and somewhat unhealthy aspect, consulted me December 1st, 1852, on account of an enlargement of the testicle. He first observed the swelling in the preceding May, and applied to Mr. Ely, a surgeon, in Rochester, shortly afterwards. Notwithstanding the remedies employed, including mercury and iodine, the testicle steadily increased in size, and when I examined it, was more than four times the natural size. The enlargement was nearly uniform, and chiefly of a solid character. Feeling fluctuation I introduced an exploring trocar, and removed from the tunica vaginalis about two ounces of bloody serum, which formed a thin layer around the diseased gland. There was no disease of the spermatic cord, and no perceptible enlargement of the inguinal and lumbar glands. Having no doubt that the disease was either carcinomatous or cystic tumour of the testicle, I recommended its removal, and performed the operation on the 8th inst. The patient recovered favorably, and returned into the country on the 26th, and I am informed by Mr. Ely, that up to the present time he has continued in good health.

On making a section of the tumour, I found a thin layer of the brown tubular structure of the testicle spread over a part of the surface, just beneath the tunica vaginalis reflexa; but the mass was principally composed of a multitude of small cysts varying in size, and in the nature of their contents. Some of the larger cysts measured half an inch in diameter, but the majority were much smaller, and many were no larger than millet seeds. A great many of

[1] Author's Treatise on the Diseases of the Testis, p. 361.

the cysts contained a transparent limpid fluid, others a bloody fluid, a few coagulated blood, and several a solid whitish opaque matter. The cysts were imbedded in fibrous tissue, which was particularly dense towards the centre of the growth. The appearances presented by a section of the tumour, are figured in Plate 1, but the cysts represented are less numerous than in the specimen.

Thin slices of the tumour being examined in the microscope, the origin of the cysts in a dilatation of the tubes was clearly made out. Thus in some specimens (figs. A, B, Plate 2), a tube could be traced to a termination in a dilated pouch. In others, a cyst appeared to arise from a lateral dilatation of a columnar tube, or at the extremity of a loup, as in fig. C, whilst in others, the dilatation appeared to be uniform, as in fig. D. These dilated tubes and cysts were lined by a tesselated epithelium, and many of them contained a dark granular matter, (fig. c.) The opaque whitish substance found in several of the larger cysts, consisted of a mass of modified tesselated epithelial scales (fig. 2). The cysts containing this white matter were firmer and denser than the others. No spermatozoa were detected in any of the cysts or morbid tubes.

This tumour must be regarded as the ordinary cystic disease, and the minute examination fully establishes the origin of the cysts in a morbid condition of the ducts. The circumstance of the healthy tubular structure being found external to the morbid growth, or extended over its surface, an arrangement which I had long ago remarked as occurring in the cystic testicle, shows that the ducts affected are not the tubuli seminiferi. If the latter were the seat of the disease, we should expect such of the tubes as remained sound to be pushed to one side, or at any rate near or mixed up with the diseased ducts, and not spread over the surface and distinctly separated from the morbid growth. Nor can the diseased ducts be those of the epididymis. In the examination of several cystic tumours of the testicle, I have found this part quite unaffected; whilst in others, it has been wasted, and lost in the morbid mass. If the disease

sprang from the tubes of the epididymis, the glandular structure of the body of the gland, if not destroyed by pressure, would certainly be found in a mass, enclosed in its own tunics, distinct from the morbid growth, and not spread over its surface.

It being clear that neither the tubuli seminiferi nor the ducts of the epididymis are the tubes which undergo these changes, constituting the cystic disease, its seat may be considered as conclusively traced to the ducts of the rete testis. Why these ducts alone are subject to the morbid change, I confess my inability to explain.

In some specimens of cystic disease in which the cysts had attained rather a large size, I have noticed growths springing apparently from the walls, and occupying more or less of the cavity of the cysts. Some of them were of a polypus form, with a narrow neck; others, of a lobular shape. In external appearance they resembled very much the intra-cystic bodies, often observed in cystic tumours of the breast. In my work on the Testis (p. 362), a specimen in the Hunterian Museum (No. 2390), showing these lobulated growths, is figured. It was removed by Mr. Liston, from a man thirty-three years of age. There was no return of the disease after the operation. On a recent examination of the growths in this specimen, which Mr. Quekett made at my request, they were found to possess a cellular structure, and to be covered on the surface with cylindrical epithelium, like that covering the villi of the intestine.

In the examination of specimens of cystic testicle, I have frequently remarked the occurrence of small masses of enchondroma mixed up with the the morbid growth, and in my article on the Morbid Anatomy of the Testicle, contained in the 'Cyclopædia of Anatomy,' the little pearly-looking masses are represented in a woodcut. In describing them, I state "that they appeared to be contained within the cysts, but I believe that they are developed externally." It is clear, however, from recent observations, that enchondroma is originally formed within the tubes and

their cystic dilatation. I have been favoured with an opportunity of examining a remarkably fine specimen of enchondromatous testicle, removed by Mr. Hancock, which was recently exhibited at the Pathological Society. The tumour had attained so great a size, as to weigh 4 lbs. 6 oz. Mr. Quekett and Mr. Hogg, in the examination of this specimen, distinctly recognised the dilated tubes as the sites of the enchondromatous masses; and elongated masses of cartilage extracted from some of the tubes, are represented in Plate 3. In other specimens of cystic disease with enchondroma, which I have examined with Mr. Quekett, the intra-tubular development of the cartilage was equally manifest. The Hunterian Museum contains a large cystic testicle, which was presented to the college by Sir S. L. Hammick. It consists of a multitude of cysts, surrounded by fibro-areolar tissue, and numerous masses of enchondroma within cysts or dilated tubes. The cartilage occurs in elongated portions resembling those already described, and are easily detached from the cysts enclosing them. Many of the larger cysts are also filled with a whitish substance, composed of a modified tesselated epithelium, similar to the whitish matter observed in the cysts of the testicle, in the case described in this paper.

The minute examination of these cystic tumours shows the non-malignant character of the disease, which, moreover, is fully confirmed by the accounts of those cases in which the history has been preserved, patients having lived many years after the excision of the organ, and died of a different disease. This is a point of considerable practical importance, for it enables the surgeon, after a minute inspection of a cystic tumour of the testicle which has been removed by operation, to assure his patient of his permanent recovery, and immunity from all risk of a relapse.

Cases, however, have occurred which, nevertheless, would strongly tend to shake our confidence in the above conclusion. Some years ago a medical friend, æt. 32, was attacked with disease of the testicle. It continued to increase in size, and at the end of eighteen months was excised by Mr.

Luke. On a cursory examination of the tumour, I found it to exhibit the ordinary appearances of cystic disease, blood being, however, extravasated in two or three places, which was attributed to some exploratory punctures made previous to the operation. The patient never regained his health, but remained cachectic. In about six months he suffered from hæmoptysis, which was followed by attacks of severe lumbar pain, and subsequently the liver enlarged to a great size. He died eighteen months after the operation. On examination of the body, masses of medullary cancer were found in the lumbar glands, lungs, and liver.

In a visit which I paid several years ago to the Museum of St. George's Hospital, Mr. Cæsar Hawkins showed me some preparations in his collection, of cystic testicles which had been removed by operation, the patients having died within two years afterwards of internal tumours, and he expressed to me his opinion that this disease of the testicle was a malignant affection. In pursuing the investigations which form the subject of this paper, it appeared important that the nature of the morbid change in these cases should be clearly ascertained, and with the sanction of Mr. Hawkins, and the assistance of the able curator, Mr. Gray, I have recently made a careful examination of the preparations alluded to.

Two specimens were shown me, one marked V 18, entitled in Mr. Hawkins's catalogue, "Fungus hæmatodes of the testis." It appeared that a small lump was removed from the epididymis, and that three or four years afterwards the testicle, which had grown to the size of a melon, was excised. The operation was performed in October, 1834. In November, 1835, the patient was attacked with disease of the lungs, and with pain in the back, and, after expectorating blood, died in the February following. The abdomen contained large masses of diseased glands, and the lungs and liver were full of fungus hæmatodes tumours, which were dark and bloody. The diseased testicle is stated to consist of a great number of cysts containing fluid, for the most part transparent, but in some dark coloured. The

bulk of the tumour was formed of thick white matter between the cysts, hard and firm in consistence, and in some places, as if filling cysts, were portions of coagulated blood.

This preparation exhibited the ordinary character of the cystic disease. I observed two or three small portions of enchondroma in the substance of the tumour, and the tubuli testis were spread out in a thin layer over its upper part, being otherwise in a healthy condition, and altogether distinct from the cystic disease. The deposits of blood were, however, more numerous than is commonly met with in cystic disease.

The second preparation, marked V 20, and also entitled "Fungus hæmatodes of the testis," was removed by Mr. Babington seventeen months before the patient's death. A tumour appeared in the neck about a twelvemonth after the operation, and another in the abdomen four months before his death. The disease in the abdomen consisted of a mass of globular tumours on the right lumbar region, (the side from which the testicle had been removed,) extending up to the diaphragm, and surrounding the aorta. There was similar disease in the omentum. The greater part consisted of a number of cysts of various colours and consistence, but many, especially about the stomach and root of the mesentery, contained a milky fluid like chyle. Others were more solid and pulpy, and of the usual appearance of fungus hæmatodes. The tumours in the neck had a similar appearance.

The cysts in this preparation were of remarkably large size, larger, indeed, than I have observed in any other specimen of cystic disease of this gland. The intervening tissue was very firm, and there were a few deposits of blood, and of a soft granular substance.

The soft matter from the cysts of both these tumours, when submitted to microscopic examination, appeared to consist of a mass of nucleated cancer cells. Some of them contained numerous dark granules, and where the diseased mass was the softest, the granules were more abundant than the cells, the cell walls in these instances having been most

probably destroyed. In some of the masses portions of ducts filled with cells might be observed. No epithelial scales could be detected in either of the specimens.

In describing a malignant form of cystic disease of the testicle, I do not comprise cases of encephaloid cancer of the organ in which two or three cysts may be found mixed up with the cancerous disease, but tumours the great mass of which is composed of cysts of various sizes. It seems probable, however, that in the early stage of this form of the disease, the cystic structure prevails, but that at a later period the cysts become destroyed by the rapid growth of carcinomatous tissue. This had probably occurred in a specimen in the Hunterian Collection (No. 2416). It is a section of a large tumour of the testicle, the upper part of which is composed of a multitude of small cysts, whilst the remainder exhibits the usual appearances of medullary cancer, of which the patient died a few weeks after the removal of the tumour by Mr. Guthrie. There can be little doubt that encephaloid cancer commences commonly, if not always, in the rete testis, and in specimens where the disease is not too far advanced, the tubuli seminiferi may be found, as in the cystic testicle, extended in a layer over the tumour. This I have observed in several instances.

The facts here adduced seem sufficient to show, that cystic disease occurs in the testicle in two forms, a malignant and non-malignant, the former being, I believe, by far the most rare. The presence in the cysts of tesselated epithelium, will indicate the character of the non-malignant, and the presence of nucleated cancer-cells the nature of the malignant, and if these observations be confirmed, we shall thus possess the means of determining a most important distinction, illustrating the valuable aid which the microscope is capable of affording in solving the difficulties of practice.

The following are the propositions resulting from the preceding inquiries:

1. Cystic disease of the testicle occurs in two forms, an innocent and a malignant.

2. Both forms are the result of morbid changes in the

ducts of the rete testis, this part of the gland being the sole seat of the disease.

3. The innocent form of the cystic disease is characterised by the presence of tesselated epithelium in the cysts.

4. The malignant form is characterised by the presence of nucleated cancer cells in the cysts.

5. Enchondroma occurs in both forms of the cystic disease, and almost constantly in old cases of the innocent, the cartilage being developed within dilated tubes.

ADDITIONAL EXPERIMENTS ON THE EXCITABILITY

OF

PARALYSED AND HEALTHY LIMBS

BY THE GALVANIC CURRENT.

BY

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Received June 28th.—Read June 28th, 1853.

In the summer of 1847 I ventured to submit to this Society the results of some experiments made with the view of ascertaining whether limbs paralysed in consequence of the removal of the brain's influence upon them were more excitable by the galvanic stimulus than healthy limbs whose muscles still remained in connection with the centre of volition.

In pursuing this subject subsequently, as opportunities offered, I found that my observations justified my adhering to the general views expressed on that occasion as to the *modus operandi* of the galvanic current when transmitted through the limbs, and furnished me with some additional facts, which I am desirous of laying before the Society as a sequel to my paper already referred to.

I shall first give an account of the results of my observations on the transmission of the galvanic current through the limbs of healthy subjects, both in man and the lower animals.

A Cruickshank's battery of fifty pairs of plates, each plate being about two inches square, charged with dilute sulphuric

acid, was employed for these experiments. The current was transmitted through the arms by connecting the poles of the battery with basins of water, in which the hands were immersed.

Twelve students of King's College Hospital kindly submitted to these trials, all apparently in robust health, and I was myself the subject of the same experiments.

It is evident from the arrangement adopted, that the current as transmitted through the arms, was direct in one arm, and inverse in the other.[1]

The following results were obtained from these experiments.

1. That the obvious physiological effect was produced only on completing or on interrupting the galvanic circuit through the limbs.

2. That a greater effect, *i. e.* more vigorous contractions, followed the completion of the circuit than its interruption.

3. That in every instance the completion or interruption of the direct current, produced more vigorous contractions than the completion or interruption of the inverse current.

The greater influence of the direct current in producing contractions was so marked, that it was easy, by observing the action of the arms, to tell the direction of the current, it being always direct in the limb in which the contractions were most active and vigorous. And each individual who submitted to the experiments readily perceived the difference in his own person, from being conscious of more lively contractions in one limb than the other.

These phenomena became still more striking, on our adopting an arrangement by which the direction of the current through the limbs could be instantaneously reversed, making it, at the same moment, inverse where it had previously been direct, and *vice versâ*. The more active contractions of the direct current, in the most striking way, seemed, as it were, to change sides with the change of direction of the current,—this being equally obvious in

[1] The current is *direct* when it passes from centre to periphery, *inverse* when it passes from periphery to centre.

the change produced in one's own sensations as the current became reversed in each limb.

4. A smaller number of plates was capable of exciting contractions by the direct current than by the inverse, thus affording a further illustration of the greater energy of the former current than of the latter.

When, by various contrivances, the current was made to pass in the same direction in both limbs at the same moment, no difference was observable in the relative power of the contractions of each limb, but it was still obvious that the contractions excited by the direct current were more vigorous than those excited by the inverse. The simplest mode of subjecting both limbs to the action of a current, which shall have the same direction in each, is by immersing the feet in one basin and the hands in another; thus, the former will be connected with one pole of the battery, and the latter with the other, the direction of the current in each pair of limbs being indicated by the pole with which they are connected.

The foregoing experiments were made with the galvanic trough. It has been asserted that the physiological action of the coil machine and of the magneto-electric machine, was not only more intense than that of the galvanic trough, but that it was also so different, that the use of the former instruments would lead to results quite opposite to those obtained by the latter.

The latter part of this statement is so much at variance with the results of my own experiments with the coil machine, and the magneto-electric apparatus, and also so inconsistent with the most reasonable theory of the mode of generation of the galvanic force by them, that I determined to try their influence upon a number of healthy persons, in the same way as I had tried the influence of the galvanic trough.

The results of these trials were exactly the same as those obtained from the experiments with the galvanic trough, *i. e.*, the contractions were distinctly greater in the limb in which the current was direct. And by changing the direction of the current in each limb, making it direct

where it had been inverse, and *vice versâ*, the contractions always became more intense when the current was made to take the direct course.

In short, these experiments showed that the physiological effects of the coil machine and of the magneto-electric machine were precisely the same as those of the galvanic trough. Both these instruments are capable of producing contractions of the utmost intensity, but they are also extremely manageable. The coil machine readily permits its intensity to be regulated, for by varying the quantity of the inducteous material its power may be made to range from almost zero up to such a degree as to excite violent tetanic convulsions and the most horrible pain.

I must leave it to others to explain how, under similar circumstances and in the same individual, *opposite* physiological effects may be produced with the galvanic trough and with the coil machine. Perhaps in the experiments which yielded these opposite results, the importance of attending to the direction of the current was overlooked.

The facts above detailed have a very important significance. They denote, not only that in comparing the excitability of one limb under the influence of the galvanic current with that of its fellow, due care must be taken to compare them under similar states of galvanic influence, —*i. e.*, with the current taking the same direction in each,— but likewise that in exciting contractions the galvanic current acts primarily upon the nerves, and through them upon the muscles.

This latter conclusion, to which I had already been led by the facts and reasonings contained in my former paper on this subject, receives strong confirmation from other circumstances.

1. The galvanic current, when applied directly to a muscle, excites it to contract with just the same intensity, whatever direction it may take.

2. If the nerves be carefully removed from the limbs of an animal just killed, the galvanic current will excite contractions on both sides, nor can any difference be observed

between the contractions of the limb in which the current is inverse and those of the limb in which the current is direct.

3. After the nerves have been removed, the limbs no longer exhibit those differences in the influence of the inverse and direct current first noticed by the Italian *physiciens*, such as the excitation of contractions on *completing* the direct current, or *interrupting* the inverse.

4. After the nerves have been removed, the excitability of the limbs by the current ceases in each at the same moment.

5. The removal of the nerves prevents the development of that remarkable phenomenon,—the sudden assumption, on breaking the circuit, of the tetanoid state by the muscles of a limb through which the inverse current has passed for some time without interruption.

I shall now proceed to narrate a few experiments made upon paralytic patients, in which greater precautions were taken than in my former experiments, both with reference to the direction of the current and to other points, which will appear in the account of each experiment.

Case I.—*April* 14, 1848. My friend Dr. Walshe, of University College Hospital, kindly permitted me to compare the excitability to galvanism of the healthy and paralysed limbs in a patient who was suffering from hemiplegia, and he also lent me his valuable assistance in the experiments.

The patient was an old woman, who had suddenly become completely paralysed on the right side; the muscles of that side were perfectly relaxed, and presented not the slightest rigidity: they were softer than those of the sound side, and appeared to have experienced a slight degree of wasting. The seizure was not preceded by any premonitory symptoms.

The first trials were made with a Cruickshank's battery. The current was passed only through the upper extremities, as it was found inconvenient to include the lower extremities.

I shall give the results in the following tabular form,

just as the notes were taken by Dr. Walshe, at the bedside of the patient:

1. With thirty pairs of plates.
   a. Current direct in paralytic limb—moderate contractions of the biceps; *none*, of the muscles of the fore-arm.
   Current inverse in the sound limb—strong sharp contractions of the biceps; strong contractions of the muscles of the fore-arm; slight jerking of the limb; contractions of the fingers.
   b. The current was now reversed, retaining the same number of plates.
   Current inverse in paralytic limb—very faint contractions of the biceps; *none* in the fore-arm.
   Current direct in sound limb—*powerful contractions* of all the muscles.
2. With twenty pairs of plates.
   a. Current direct in paralytic limb; very slight contraction of the biceps only.
   Current inverse in sound limb; as much (if not more) contractions of the biceps, and also distinct action of the muscles of the fore-arm.
   b. Current inverse in paralytic limb. *No action.*
   Current direct in sound limb; strong action.

Thus it appeared that with twenty pairs of plates the *direct* current produced strong action in the healthy limb; and caused slight contractions of the biceps muscle only in the paralytic limb.

3. With ten pairs of plates. No action in either limb.
4. With the entire battery (50 pairs of plates).
   a. Current direct in paralytic limb—free and strong contraction of the biceps—but *no contractions* of the muscles of the fore-arm.

b. Current inverse in sound limb—free action of the biceps—the contractions being quite as strong, but not stronger, than those of the paralysed limb; *intense contractions* of the muscles of the fore-arm.

Thus, with the whole force of a battery of fifty pairs of plates, and the advantage of a direct current, the muscles of the paralysed fore-arm could not be excited to action; while even with the inverse current *intense* action was excited in the healthy limb.

We next used the coil machine: the result of the influence of this instrument was, that with the current direct in the paralysed limb there was moderate action of the biceps of the paralytic arm, but none of the muscles of the fore-arm, whilst the muscles of the sound limb contracted freely. On reversing the current, the muscles of the healthy limb became much more active in their contractions, and the biceps of the paralysed limb contracted only very feebly.

The influence of the coil-machine, therefore, produced results in exact correspondence with those obtained from the galvanic battery.

The patient who was the subject of these experiments died of pneumonia very shortly afterwards. On post-mortem examination, there was found softening of the left corpus striatum, with spots of yellowish discoloration, probably from small hæmorrhage. No trace of pus or other inflammatory product could be discovered.

Case II.—June 19, 1848. Jane Williams, æt. 36, a patient in Guy's Hospital, under Dr. Addison, who with Dr. Gull witnessed the experiments. There was hemiplegia of the left side; the paralysis was complete, and the muscles quite flaccid.

The experiments were made with the Wollaston's trough, and on the upper extremities only. When thirteen pairs of plates were employed, no contractions were excited on either side, whatever the direction of the current.

With seventeen plates, no contractions were excited in the paralysed limb with either the direct or the inverse current, while slight ones were produced in the healthy one, chiefly in the fore-arm. These were much more distinct when the current was direct than when it was inverse.

With twenty-five pairs of plates distinct and strong contractions were produced in the sound limb, slight ones in the paralysed, which were confined to the fore-arm. The difference in the intensity of the contractions was very great, whatever the direction of the current.

With thirty-three pairs of plates, and afterwards with fifty, the same contractions became general in both limbs, but with a marked difference of intensity in favour of the healthy limb, whatever was the direction of the current. This difference was more manifest when the current was direct in the healthy limb, less so when direct in the paralysed limb.

The lesion in this case, as I learned afterwards from Dr. Gull, consisted in very extensive colourless softening of the white substance of the right hemisphere.

Case III.—Catherine Williams, æt. 50, admitted into King's College Hospital. Two days before her admission, while sitting at breakfast, she suddenly lost the use of the left arm and side of the face, and in the evening of the same day the left leg became paralysed. The attack was not accompanied by any loss or impairment of consciousness. On admission, she was found completely hemiplegic on the left side, the facial palsy being complete, and the tongue, when protruded, deviating to the left side. The paralysed muscles were very much relaxed; there was not the slightest appearance of rigidity nor the least resistance, when extension or flexion of either limb was effected.

The trials of galvanism were made with the trough and with the coil machine, both of which yielded similar results. Whatever the direction of the current, very faint contractions were excited on the paralysed side—whilst those on the sound side were distinct, although not very strong; they were more distinct under the influence of the direct current.

This woman was in a state of great debility, and did not long survive her admission into the hospital. After death, the lesion was found to be extensive softening, with loss of substance of the right corpus striatum, but without any sign of an attempt at cicatrisation.

Case iv.—Thomas Hardwick, æt. 49, a smith, admitted into King's College Hospital, August, 1848. In this patient the hemiplegia was on the right side, the power of motion was not completely lost; more so in the arm than in the leg; there was some loss of sensation in the arm. The muscles of the paralysed limbs were firm and rigid, especially those of the arm. This rigidity was sufficient to give a condition of semi-flexion to both arm and leg, especially the former, which was most conspicuous at the elbow and in the fingers.

This patient was galvanised in the usual way, with more effect on the *paralysed* than on the *sound limbs*, which was very obvious in the early part of his stay in the hospital. After some time, the muscles of the palsied limb became less rigid, and in proportion as the rigidity became less the influence of the galvanism diminished likewise.

In examining the brain of this patient after death, it appeared that the lesion consisted of inflammatory disease of the pia mater, compressing and irritating the optic thalamus of the left side. An irritating lesion of this kind explained the rigid or spastic condition of the muscles of the paralysed limb. This was due to an exalted polarity of the nerves, which kept the muscles in this firmly contracted or spasmodic state, and to the same state of nerve was to be attributed the increased excitability of the muscles of that side to the galvanic stimulus.

Case v.—Elizabeth Clarke, æt. 63. (Case Book, vol. xxiii, p. 163.) This woman was a housekeeper at one of the west-end clubs. She was admitted on the 1st of May, 1848, semi-conscious, having just had a paralytic stroke, which induced complete hemiplegia of the right side. The muscles of the arm and leg were perfectly lax.

The first trial with galvanism was made on the 3d of May, two days after her admission, with the Cruickshank's battery. With twenty-five plates, and a direct current in the right or paralysed arm, very feeble contractions were excited in that side; distinct ones in the left, or sound side, although in it the current was inverse. On reversing the current, the contractions became very vigorous in the sound arm, and were scarcely perceptible in the paralysed.

With fifty plates, very distinct contractions were excited in both arms; those of the sound side, whether with the inverse or the direct current, being always obviously the stronger.

A second trial with the galvanic current was made on the 15th. Her mental functions had much recovered, but the palsy remained in *statu quo*. The results of this trial were precisely the same as regards the *comparative* effects of the galvanic current on the two sides; the only change being that a little more power of contraction was exhibited on both sides, which was probably due to an improved state of the patient's general health. On the 17th, these experiments were repeated with the same results.

On this occasion, an arrangement was adopted, by which the current was divided, and made to pass down each arm as a direct current. Thus each arm was, at the same time, similarly affected by the galvanism. The contractions were invariably stronger in the sound than in the paralysed limb. When the current was inverse, the contractions on both sides were very feeble, but more pain was excited, and greatest muscular action in the sound limb.

The palsy in this case was caused, in all probability, by white softening of the left side of the brain, due to defective nutrition, and possibly there might have been slight hæmorrhage into the substance of the brain. The patient remained two months in the hospital, and partially recovered the power of the limbs.

Case VI.—John Shea, æt. 44, admitted April 4, 1849, a bricklayer, and of intemperate habits. Going down by

railway to Sydenham, on the 2d of April, he had a fit in the carriage, and was found lying on the floor in a state of insensibility. On recovery, he was found completely paralysed on the right side. There was complete relaxation of the muscles, except the biceps. The right side of the face was paralysed in a very marked manner, and the tongue, on being protruded, deviated to the right; speech was impaired. The lethargy consequent upon the attack did not go off for some days. The first trial with galvanism took place on the 5th of April. The Cruickshank's trough was used as in the other experiments.

With five plates, no contractions were excited in either arm.

With twelve plates, there were slight contractions in the left fore-arm (the sound side), none in the right (the paralysed).

With fifteen plates, contractions in both fore-arms, but decidedly weaker in the right.

With twenty plates, manifest contractions in both, but distinctly weaker in the right. When the arms were loosely suspended in a basin, the right or palsied arm was most jerked, evidently in consequence of the lax state of the muscles and the absence of due antagonism.

With twenty-five plates, vigorous contractions, but the superiority of the left over the right was still very manifest.

In these experiments the current was always direct in both arms. The patient's hands were immersed in one basin, and his feet in the other, and the positive wire was connected with that in which the legs were placed.

A second trial with the galvanism took place on the 26th. No improvement had taken place in the motor power of the paralysed side; but the sentient power, which, after he had rallied from the shock of the palsy-stroke, we found had been very much impaired, had now nearly recovered itself. The condition of the muscles was the same.

On this occasion the results of the experiments were precisely the same as before.

In this case I have no doubt the lesion was a clot, which involved a considerable portion of the left corpus stria-

tum. The patient frequently visits the hospital, and I have seen him within the last few days (May, 1853). He has not recovered the palsy, excepting, in a slight degree, that of the leg. The muscles of the fore-arm have become wasted and contracted, causing permanent flexion of the fingers, and of the fore-arm upon the arm, a condition which, in my opinion, is apt to follow the absorption of a clot, and the contraction and cicatrisation of the torn brain-substance.

CASE VII.—John Liftin, æt. 55, admitted April 11th, 1850. On Easter-day (April 9th), at half-past ten o'clock in the morning, whilst walking across the room, he dropped a candlestick which he was carrying in his hand, and his wife then found that he had lost the use of his right side; he soon became very lethargic, with stertorous breathing; in this state he continued for some days. On coming out of this lethargy, he was completely paralysed, with relaxed muscles on the right side, the face, tongue, arm, and leg being affected, and his powers of articulation were very much impaired. Sensibility was not injured; on the contrary, pinching the skin occasioned more pain in the paralysed than in the sound limb.

The galvanism was tried first on the 14th of April.

With nine plates, the palsied side having the advantage of the direct current, much the strongest contractions were excited on the sound side. On inverting the current, very strong contractions were excited in the sound limb; those in the paralysed were very slight.

With eighteen plates, the current being direct in both arms, the contractions were strong and distinct in the sound side, very feeble in the paralysed. They were also very feeble in the legs, in which the current was inverse.

This patient remained in the hospital many weeks. He recovered in the course of the first four weeks a certain amount of power in the leg, so as to enable him to walk with the characteristic hemiplegic jerk, but gained no power in the arm.

On the 10th of June, a second trial was made with gal-

vanism. The current was made to pass through both arms and legs, so that it was direct or inverse in both arms, or in both legs, at the same time.

When the current was direct in both arms, decided contractions were excited, varying in force with the number of plates used, but with a very marked difference in favour of the sound arm. Analogous phenomena were observed under the influence of the inverse current; but the contractions in both upper extremities were decidedly less, although those of the sound arm predominated distinctly over those of the paralysed.

This, like the preceding case, was, in all probability, one of apoplexy, leaving a clot in the left hemisphere of the brain; the patient had made very little advance towards recovery when he left the Hospital, and probably would never perfectly recover the use of the right side. He has not since been heard of.

In the next case the influence of the coil machine was first tried, and afterwards that of the battery.

Case viii.—William Ware, æt. 30, a ploughman, (Case Book, vol. xxvi, A;) fifteen weeks before his admission he became suddenly paralysed on the right side in the night, having for some days previously experienced a numbness in the arm and leg of that side. On awaking in the morning he found himself in this hemiplegic state, but speedily lapsed into a state of unconsciousness, which lasted, more or less, for three weeks. As his intelligence recovered, he seemed to gain a little power over the affected side. He yawned frequently, and crying was excited on the slightest emotion.

On admission there was hemiplegia of the right side, the face and tongue being affected as well as the arm and leg. Some power had been recovered, and he could flex the fingers so as to grasp feebly. The leg had recovered nearly completely. The muscles of the arm were quite flaccid, and were but slightly wasted. Those of the leg had not yet recovered their usual size.

The first trial of galvanism was made with the coil machine.

When the current was direct in the *paralysed* side, and inverse in the sound, the contractions were equal in both arms.

But when the current was inverse in the paralysed and direct in the sound, the contractions of the former were feeble—those of the sound limb being strong and even violent.

The direct current caused greater contractions in the sound limb, than the same current in the paralysed.

But the inverse current caused distinctly stronger contractions in the paralysed side than the same current in the sound.

Two days after the above experiments a second trial was made, and now the Cruickshank's battery was used.

The direct current produced the greatest contractions through whichever limb it was directed, and those of the sound limb were distinctly greater than those of the paralysed. But, on transmitting a divided inverse current through both arms, the muscles of the paralysed arm were decidedly more affected than those of the sound limb.

After the lapse of a fortnight the leg had nearly completely recovered, and additional power had been gained in the arm. A third trial with the Cruickshank's battery showed that whether with the direct or inverse current the contractions, as well in the arms as in the legs, were distinctly greater on the sound than on the paralysed side. On this occasion the greater contractions of the paralysed arm under the inverse current were not observed.

This phenomenon of the greater contraction of the paralysed than of the sound arm under the inverse current, seemed to me attributable to some altered conditions of the nerves of the former limb, due, perhaps, to the process of repair going on in the brain.

The patient caught erysipelas about three weeks after the last trial of galvanism, and quickly succumbed under it. We had thus an opportunity of ascertaining that the left

half of the left corpus striatum was broken down and excavated into a small cavity, filled by an opaque buff-coloured fluid. Several bands of fibres passed across its posterior half. All the rest of the corpus striatum was healthy. The portion immediately surrounding the cavity was a little soft, but not otherwise altered.

Case ix.—John Frost, æt. 50, admitted February 16, 1850. The attack was sudden, only a few days before admission; the patient was in a semi-comatose condition. There was complete paralysis of the right arm, with relaxation of all the muscles, except the biceps, which resisted extension of the forearm. The leg was only partially paralysed. The tongue was protruded to the left side, and the left cheek was paralysed.

The trial with galvanism was made in the usual way, giving the paralysed arm the advantage of the direct current; there was, nevertheless, a marked difference in the contractions in favour of the sound limb. The difference was much more manifest when the current was reversed, so that it became inverse in the palsied, and direct in the sound limb.

Subsequently the arrangement was adopted by which both arms became simultaneously excited by the direct or by the inverse current. The contractions in both arms were much stronger with the direct than with the inverse current, but with both, those of the sound limb were decidedly the strongest.

This patient died from the exhausting influence of diarrhœa eleven days after the attack. On examination, the lesion was found to be white softening of the left optic thalamus and the neighbouring fibres of the hemisphere, and a recent clot in the thalamus occupying its external two thirds.

Case x.—S. Jago, æt. 40, a glass-cutter, of intemperate habits. Complete hemiplegia of the right side. The attack of palsy came on suddenly after previous head symptoms,

pains in the head, failure of memory, low spirits. These symptoms dated from a fall whilst intoxicated, twelve months before the attack of palsy.

He was admitted into hospital three weeks after the palsy-stroke, with complete paralysis of the right arm and leg, the muscles being relaxed.

The only trial with galvanism was made four days after his admission. The hands were placed in one basin and the feet in another; thus the current was direct or inverse at the same time, in both arms or in both legs. With both the inverse and the direct current, much more vigorous contractions were excited in the sound arm or leg than in the paralysed.

Case xi.—J. Dowden, æt. 30, admitted June 8, 1850, for hemiplegia of the right side. The paralysis was perfect, and the muscles relaxed, but there was a tendency to flexion of the fingers, which resisted extension slightly. The attack occurred six weeks before admission; it was sudden, she fell, but retained her consciousness for a short time, but soon lapsed into a comatose state, out of which she came in three or four hours with palsy of the right side and loss of speech; she had had three attacks of rheumatic fever, and her heart was diseased, the mitral valve having been damaged.

On admission, this patient was recovering a slight amount of power in the leg and arm. There were strong reflex actions in the leg, and the arm was jerked up whenever she yawned.

The trial with galvanism was made in the usual way very soon after her admission. When the current was direct in either arm, the contractions of that arm were the more violent and the stronger. When it was direct in both arms, the contractions of the sound arm were the stronger; it was so likewise when the current was inverse in both. But, in both instances, whether with the inverse or the direct current, the difference between the contractions of the two limbs was not very great.

This woman remained in hospital about a month, and acquired some but not much additional power.

CASE XII.—For this case and the observations made upon it, I am indebted to my friend Dr. Brinton, in whose accuracy of observation the greatest reliance may be placed.

George Jenkins, æt. 30, on rising from his bed on the morning of February 23, 1852, found to his great surprise that he had entirely lost the use of his left side, so that he fell upon the floor. His speech was also impaired, but his intellect was not affected.

There was complete paralysis, with relaxed muscles of the left arm and leg, "these limbs lying like logs, incapable of the slightest obedience to the will." There was also nearly complete loss of sensation, and not the slightest appearance of reflex action could be produced.

After a few days' treatment with purgatives, and a blister to the back of the neck, galvanism was tried. The results of these trials I shall give in Dr. Brinton's words.

"The galvanic stimulus made use of was an electro-magnetic apparatus, so graduated by the bundle of wires as to give a smart but quite bearable amount of stimulus to the sound arm. On applying this to the affected side, it was at first found impossible to excite any contraction of the paralysed muscles. In about half an hour movements were gradually produced.

"On the next day, I found that there was still a marked difference between the two sides. With all gradations of shock, short of the most violent, one could plainly see, that the effect on the sound side was vastly greater than that on the other. It appeared to make little difference whether the positive or negative pole corresponded to the central extremity of the nerve along which the current was led."

In the detailed account of the case with which he has favoured me, Dr. Brinton makes the following remark, "Two circumstances," he says, "ought, I think, to be noticed in a comparison of these contractions, both of them having a tendency to represent the effect in the healthy limb as less

than what it really is. The first is the evidently more co-ordinate and general character of the contractions on the sound side; which are sometimes contrasted by small, but comparatively isolated spasms on the diseased one, better seen among the torpid and relaxed parts which surround them. The second is, that the patient sometimes controls a tolerably powerful spasm in the sound limb by a vigorous exercise of the will. The former I suspect to be very difficult of proper correction, even though the eye be assisted by the touch."

This patient gradually recovered the power of the left side, and as he advanced towards recovery, the difference in the excitability of the two sides to galvanism became less and less marked.

CASE XIII.—James Scott, æt. 53. Paralysis of the right arm, leg, and side of the face, with relaxed muscles. The paralytic seizure occurred on the 17th of November, and was not accompanied by any loss of consciousness. The trial with galvanism was not made until the 6th of December, when he had gained some power in both leg and arm. He could raise his hand to his mouth with difficulty, and bend the fingers feebly, and he could bend the knee somewhat, and raise the heel six inches from the bed, the whole limb being kept straight.

The galvanism was tried in the usual way, with Cruickshank's battery, and first with each hand in a basin with 19 plates, the current being direct in the right or paralysed arm. The right hand and arm moved more than the left, but the muscles of the left contracted with greater power, which was especially observed in the biceps, this muscle acting vigorously on the left side, while scarcely any action was observed on the paralysed or right side, notwithstanding that it had the advantage of the direct current.

When the current was inverse in the right or paralysed side, no contractions whatever could be distinguished in the right limb, either by the sight or by firmly grasping it in

different places. Vigorous contractions, however, were both seen and felt in the left limb.

The hands were now placed in one basin, and the feet in another, and forty-five plates were used.

Current direct in the arms.—The greatest motion was observable in the right arm, but the muscular contractions were firmer, and more vigorous in the left limb, both in the arm, and also in the fore-arm.

Current inverse in the arms.—The same phenomena were observed, but in a less degree.

Case xiv.—Mary Reeves, æt. 49. In this patient the paralytic attack was preceded for a period of fifteen months by pains, which for a time were regarded as rheumatic, in the right arm and leg. Four months before her admission into the hospital, in November, 1852, she was seized, while out walking, with violent pain in her right foot, which caused her to limp, and she walked home with assistance, and not without considerable difficulty. The following day the pain subsided, but the leg and arm remained partially paralysed, and this paralytic condition gradually increased during the next fortnight. She now became subject to pain in the head, chiefly in the frontal region. Early in November (the 9th), she had a fit of loss of consciousness, and since that time she has had several attacks of a similar kind, in which she has fallen with loss of consciousness, but without convulsion. The memory had been failing for some time, and speech was much impaired.

She was admitted on the 24th, in a semi-conscious state, with nearly complete hemiplegia of the right side. The muscles of the arm and leg were rigid, those of the former especially. The arm was bent across the chest, the fore-arm being flexed on the arm, and the fingers in a state of flexion. There remained slight power of moving the fingers. The sensibility seemed rather exalted, as pinching, which was scarcely felt on the sound side, caused considerable pain in the paralysed arm. Reflex actions were exalted.

The direct current, with twenty-five pairs of plates, and

with thirty-two pairs, caused distinctly more powerful contractions in the sound arm than in the paralysed, whether the experiment was tried by placing a hand in each basin, and so passing the current through the arms; or whether the current was transmitted through both legs and arms, the feet being in one basin, the hands in another.

When the current was inverse in both arms, the difference of contraction was very slight, but it appeared to two observers to be in favour of the right or paralysed arm.

It should be stated that these observations were made a fortnight after the patient's admission into the Hospital, when the rigid state of the muscles of the paralysed limbs had nearly completely subsided under treatment.

This patient died on the 12th of December, and the lesion was found to be extensive softening of the left corpus striatum, extending backwards to the mesocephale, and downwards to the crus cerebri. The posterior portion of the upper surface of the mesocephale, and the posterior part of the left optic thalamus were in a very indurated condition.

In reviewing the preceding fourteen cases, I would direct the reader's attention to the following points:

1. That of all the cases, only three (Cases IV, VIII, and XIV) exhibited under the galvanic stimulus any approach to a greater excitability of the paralysed than of the sound limb, and that in two of these (VIII and XIV) it was manifested only under the influence of the inverse current.

2. That in three of the cases (I, III, and VIII), the galvanic stimulus was applied by both the coil machine and the Cruickshank's trough, and with results precisely the same. And in one of the cases (XII) the coil machine alone was used, with results corresponding with those obtained in similar cases by the galvanic trough.

3. That in each of the three cases, in which a greater excitability existed in the paralysed limbs, the paralysing lesion in the brain was more or less of an irritative kind. In Case VIII, the irritation was probably connected with an incipient process of cicatrisation.

4. That in many of the experiments all degrees of galvanic power were used, and with no other difference than that of degree, the amount of physiological effect produced being exactly proportionate to the power of the galvanic stimulus.

The experiments which I have detailed have a two-fold bearing; first, on physiological doctrine; secondly, on practice.

As to physiological doctrine, the greater or less excitability of a muscle or set of muscles under the galvanic stimulus must depend on one of two causes, either on a *plus* or *minus* condition of the muscular force, or on a similar condition of the nervous force in the nerves which supply the muscles.

Those who maintain that the spinal cord is the source whence the muscles derive their peculiar power of contracting or their irritability, adopt the former view; and it follows, as a necessary consequence upon this, if the brain be the exhauster of this power, while the spinal cord is the magazine whence it is ever flowing to the muscles, that when the influence of the brain is removed, as in hemiplegia, the force would accumulate in the muscles, and ere long exist in them in a *plus* condition.

It was with a view to test the accuracy of such a doctrine as this, that the experiments which I formerly communicated to the Society were undertaken. It appeared to me, then, and I think so now, that if this doctrine were true, the augmented excitability of limbs paralysed by cerebral lesion ought to be so easily demonstrable, that "he who runs may read." And so, indeed, it seemed, from the experiments which were detailed on the first enunciation of this doctrine.

But the experiments, which I have instituted, show that an augmentation of the excitability of the paralysed limbs is the exception, while a diminution of it is the rule, and that every now and then we meet with instances in which the paralysed limb is wholly inexcitable, even by a powerful galvanic stimulus.

It is satisfactory to me that the results of my observations

have received ample confirmation from the similar experiments of two such competent observers as M. Brown-Sequard and M. Duchenne.

My first series of experiments was objected to, because it was affirmed (my own statement to the contrary notwithstanding, vide 'Med.-Chir. Trans.,' vol. XXX, p. 209), that I had used *only* the electro-dynamic, or the magneto-electric rotatory apparatus; and it was even asserted that *opposite* results were obtained and obtainable with either of these instruments and with the galvanic trough.

I have now shown that in healthy individuals these different instruments produce precisely the same physiological effects. And in four of the recorded cases of paralysis, the coil machine was used with results exactly corresponding with those obtained from the galvanic battery.

As regards the practical bearing of these experiments, I can only repeat that which I have stated in my former communication on this subject, namely, that the greater or less excitability of a paralysed limb to the galvanic stimulus is only an indication of the *plus* or *minus* condition of the nervous force in the nerves of that limb, and that it is sometimes of use in practise, as denoting that the paralysing lesion is, or is not, irritant, or even inflammatory.

I must also adhere to the opinion before expressed, that we gain no assistance from testing the relative excitability of the paralysed and sound limbs in the diagnosis between cerebral and spinal palsy.

By cerebral palsy, I mean that caused by disease of the encephalon, or the nervous mass within the skull; by spinal palsy, I mean that originating in disease affecting the intra-spinal nervous mass.

We will suppose a patient suffering from complete palsy of the right arm, and of that alone. The point to determine is, whether such paralysis be due to a simply local palsy, or to disease of the encephalon, or of the spinal cord. We cannot derive any assistance in the solution of this question from examining the relative excitability of the sound and of the paralysed limb: for not only may there

be, as I showed in my former paper, an augmented excitability of the muscles where the palsy is purely spinal, but in the majority of brain palsies, the excitability of the paralysed is less than of the sound limb. There is no fixed relation *as to place* between the paralysing lesion and the state of the excitability of the affected limbs; there is, however, as *to the nature* of the lesion, whether that be irritating or depressing. And a lesion of the nervous centre will influence the excitability of the limb or limbs alike, according to its nature, whether it be *intra-cranial* or *intra-spinal.*

In the repetition of experiments on this subject, I would suggest to those who might be disposed to try them, that attention should especially be paid to the state of the muscles, whether they are quite flaccid and relaxed, or whether they are rigid; and, if the latter, whether the rigidity came on soon after or at the same time with the palsy-stroke, or at a remote period? These points, as well as the state of nutrition of the muscles, should be noted in the record of the experiments. Care should also be taken to compare the action of the limbs under the same state of galvanic excitement, that is, with the galvanic current, direct or inverse in each limb. The neglect of this precaution will certainly lead to fallacious conclusions.

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ALDERSON, JAMES, M.D. F.R.S.

*Lumleian Lectures, 1852, on the Effects of Lead upon the System, and on the Mode in which Water is rendered impure by the presence of Lead, delivered at the Royal College of Physicians. 4to. London, 1852.

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*A Treatise on Corns, Bunions, and Ingrowing of the Toe-Nail; their Cause and Treatment. 8vo. London, 1852.

BABINGTON, B. G., M.D., F.R.S.

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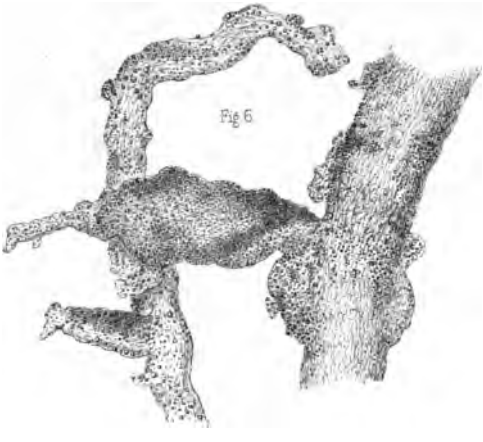
ERRATA.

Page 229, line 24, *for* Scherrer, *read* Scherer.
„ „ „ 31, *for* Scherrer, *read* Scherer.

C. AND J. ADLARD, PRINTERS, BARTHOLOMEW CLOSE.

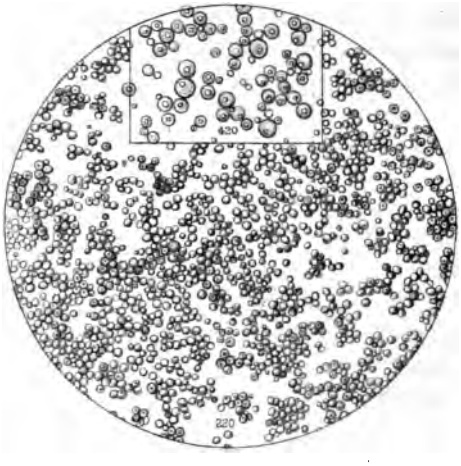
1 Villus loaded with earth and oil. 2 Villus very oily. in both the investing membrane is beginning to disintegrate. 3 Villi loaded with oil. Decidua splitting off undergoing fibrous conversion. 4 Group of villi loaded both with oil and earth: the latter disappeared on the addition of Acetic Acid.



5 *Villi from early ovum some a little oily.* 6. Villi of nonplacental chorion as they exist at the end of Pregnancy.
7 Decidual cells from early ovum. 8. Decidua from mature placenta.

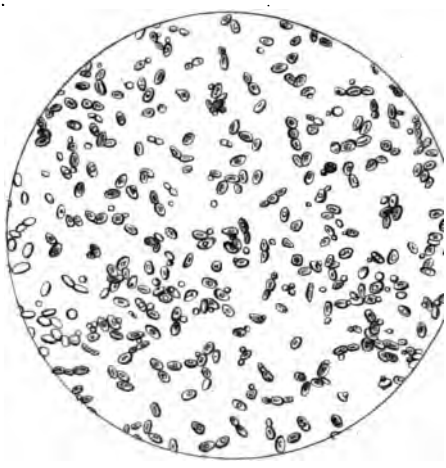
ml. & T. West, sculp.

Fig. 1.



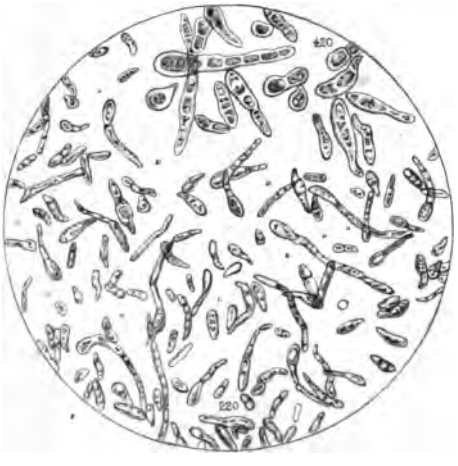
Circular Sporules of Penicilium glaucum.

Fig. 2



Oval Sporules of Penicilium glaucum 220 diam.

Fig. 3.



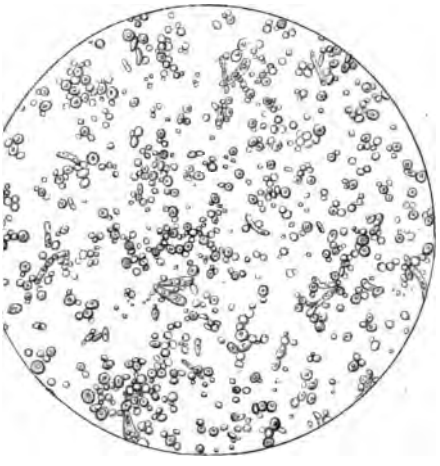
Sporules of Penicilium glaucum passing into filments

Fig. 4.

Thallus of Penicilium glaucum shewing the Vesicular Enlargements. 220 diam.

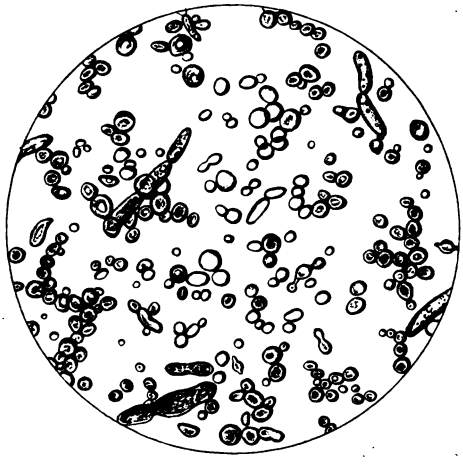
Bird & West lith 54, Easton Gardens.

Fig 1.



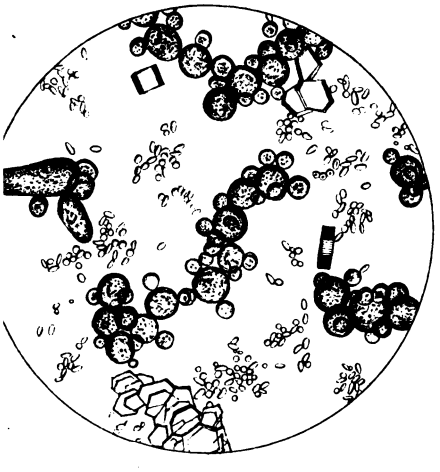
Sporules of Sugar Fungus in earliest stage of developement. 220 diam.

Fig. 2.



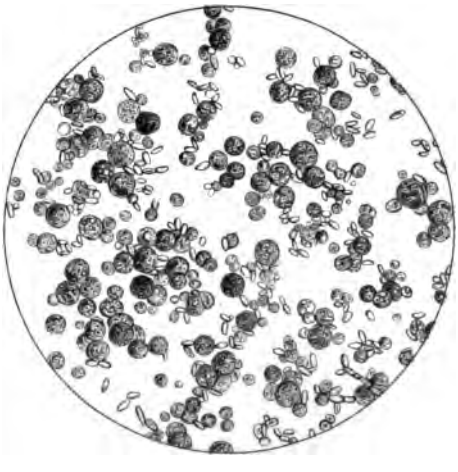
Sporules of Sugar Fungus in earliest stage of developement. 420 diam.

Fig. 3.



porules of Sugar Fungus also those of Penicilium glaucum with Crystals of Uric Acid 220 diam.

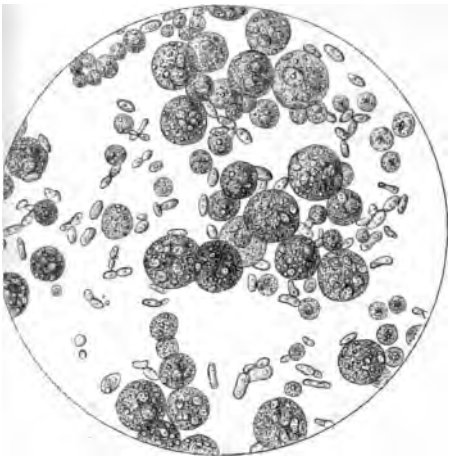
Fig. 4.



Variety of Sporules of Sugar Fungus with those of Penicilium glaucum 220 diam.

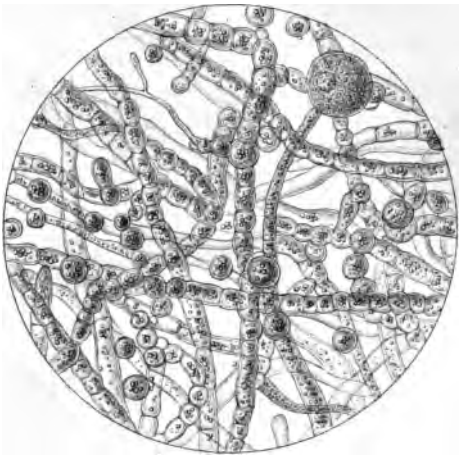
Rose & Wray, Nos. 54, Bartlett Chambers

Fig. 1.



Variety of Sporules of Sugar Fungus with many of Penicilium glaucum. 420 diam.

Fig. 2.



Thallus and Sporules of Sugar Fungus 220 diam.

Fig. 3.

Thallus and Sporules of Sugar Fungus with crystals of oxalate of lime also Vesicles of the Same 440 d

Fig 4.

Penicilium glaucum in Perfect Fructification with Filaments and Sporules of Sugar Fungus. 220 diam.

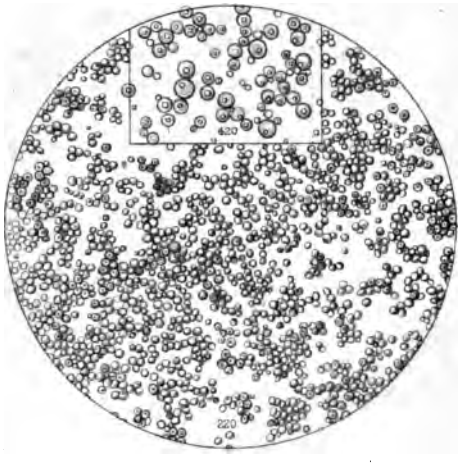
Ford & West. lith. 54 Heaton Gardens

Incipient Thallus of Penicilium glaucum with Groups of Sporules 220 diam.

Penicilium glaucum Perfect fructification 220 diam.

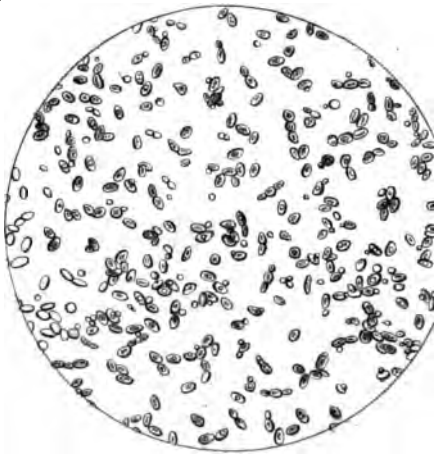
Reed & Wiese, lith. 54, Hanson, Cambridge.

Fig. 1.



Circular Sporules of Penicilium glaucum.

Fig. 2.



Oval Sporules of Penicilium glaucum 220 diam.

Fig. 3.

um glaucum passing into filments

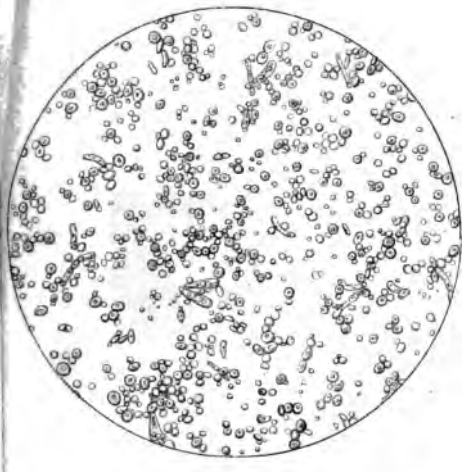
Fig. 4.

Thallus of Penicilium glaucum shewing the Vesicular Enlargements 220 diam.

Hypertrophy & Prolapse of Tongue.

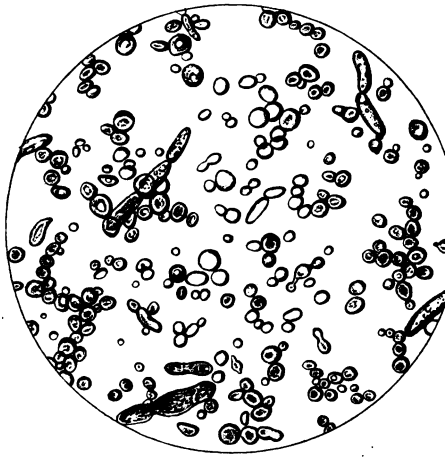
Ind. & West Ind. Sc. Medical Gazette.

Fig 1.



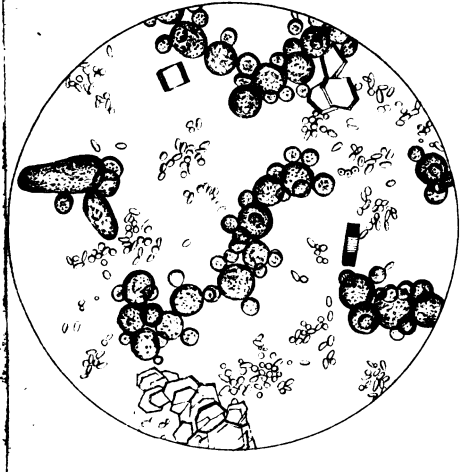
Sporules of Sugar Fungus in earliest stage of developement 220 diam.

Fig. 2.



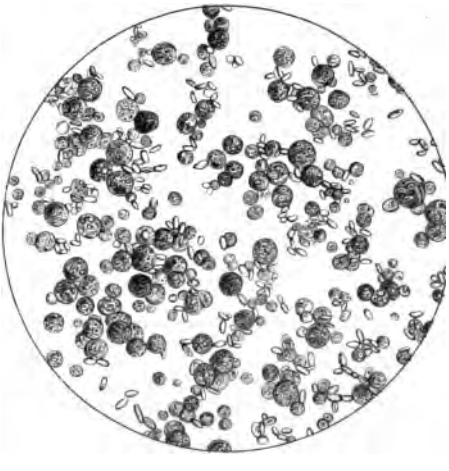
Sporules of Sugar Fungus in earliest stage of developement. 420 diam.

Fig. 3.



Sporules of Sugar Fungus also those of Penicilium glaucum with Crystals of Uric Acid 220 diam.

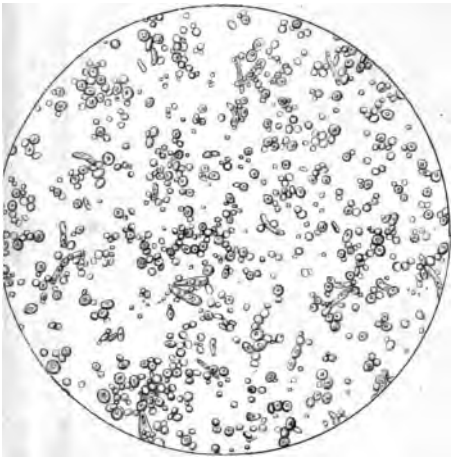
Fig. 4.



Variety of Sporules of Sugar Fungus with those of Penicilium glaucum 220 diam.

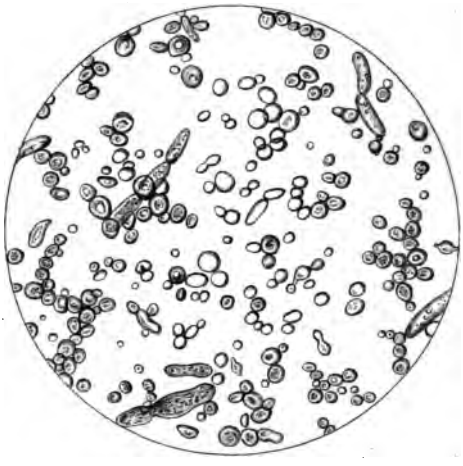
Ford & Wood, Lith. St. Patrick, Canada

Fig 1.



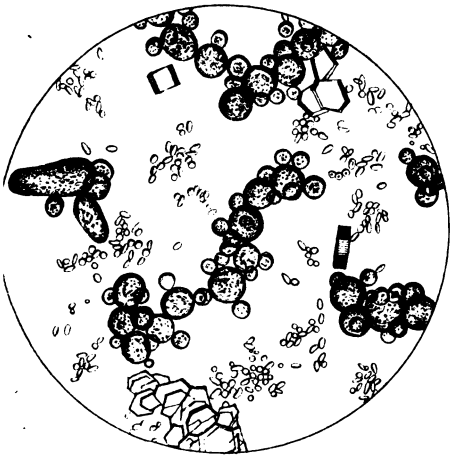
Sporules of Sugar Fungus in earliest stage of developement. 220 diam.

Fig. 2.



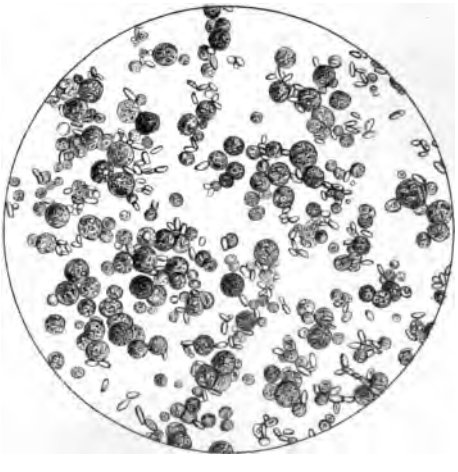
Sporules of Sugar Fungus in earliest stage of developement. 420 diam

Fig. 3.



Sporules of Sugar Fungus also those of Penicilium glaucum with Crystals of Uric Acid 220 diam.

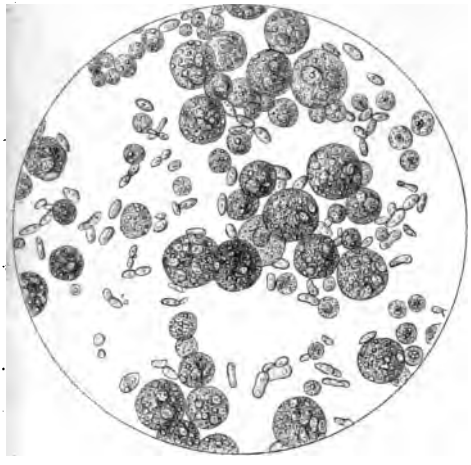
Fig. 4.



Variety of Sporules of Sugar Fungus with those of Penicilium glaucum 220 diam.

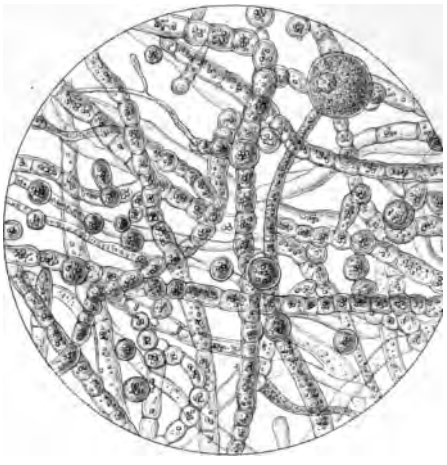
Ford & West. lith. 54, Hatton. Garden.

Fig. 1.



Variety of Sporules of Sugar Fungus with many of Penicilium glaucum. 420 diam.

Fig. 2.



Thallus and Sporules of Sugar Fungus 220 diam.

Fig. 3.

Thallus and Sporules of Sugar Fungus with crystals of oxalate of lime also Vesicles of the Plant 210 d

Fig 4.

Penicilium glaucum in Perfect Fructification with Filaments and Sporules of Sugar Fungus 220 diam.

Ford & West lith 54 Hanson Gard

Incipient Thallus of Penicilium glaucum

Penicilium glaucum Perfect fructification 220 diam.

Fig. 2.

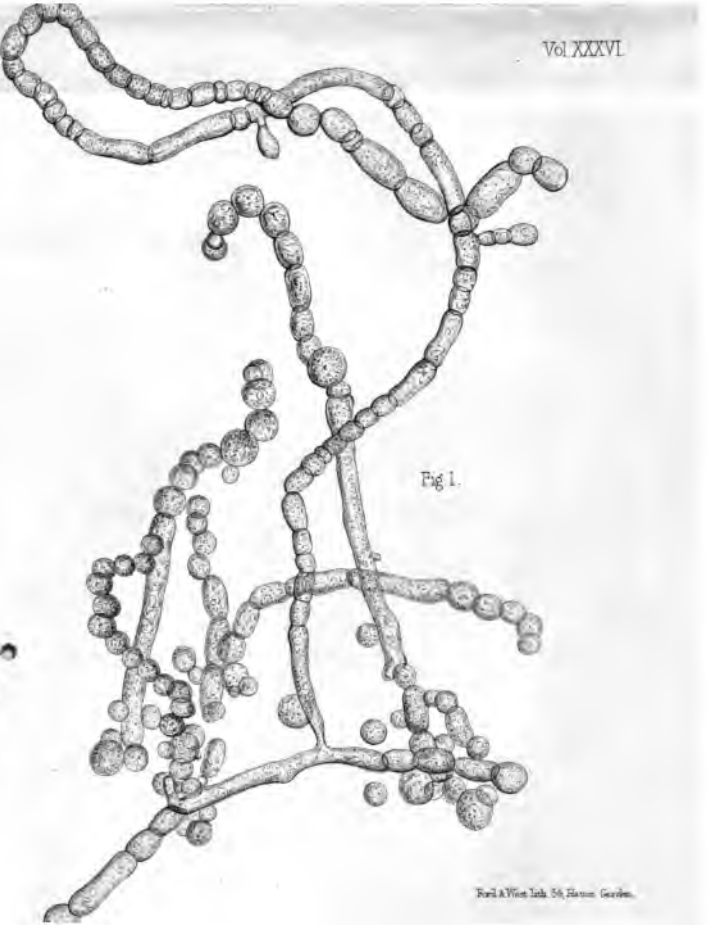


Fig. 1.

Reeve & West, Lith. 56, Hatton Garden.

Hypertrophy & Prolapse of Tongue

Ford & West Lith. to Hastings Genden

Pl. 1.
Vol. XXXVI

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Fig 1.

Accumulation of born granules and spherules of oil in dilated extremities of villi are dispersed over whole structure Nuclei of Chorion comparatively slightly affected 220 diam.

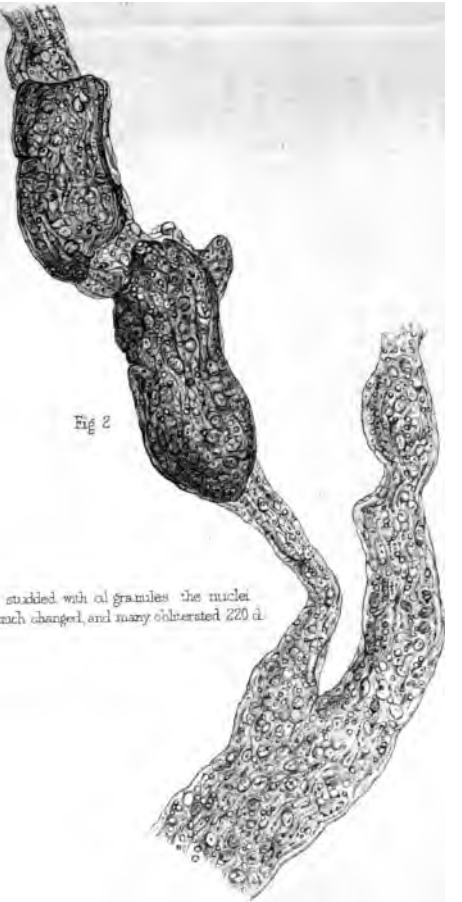
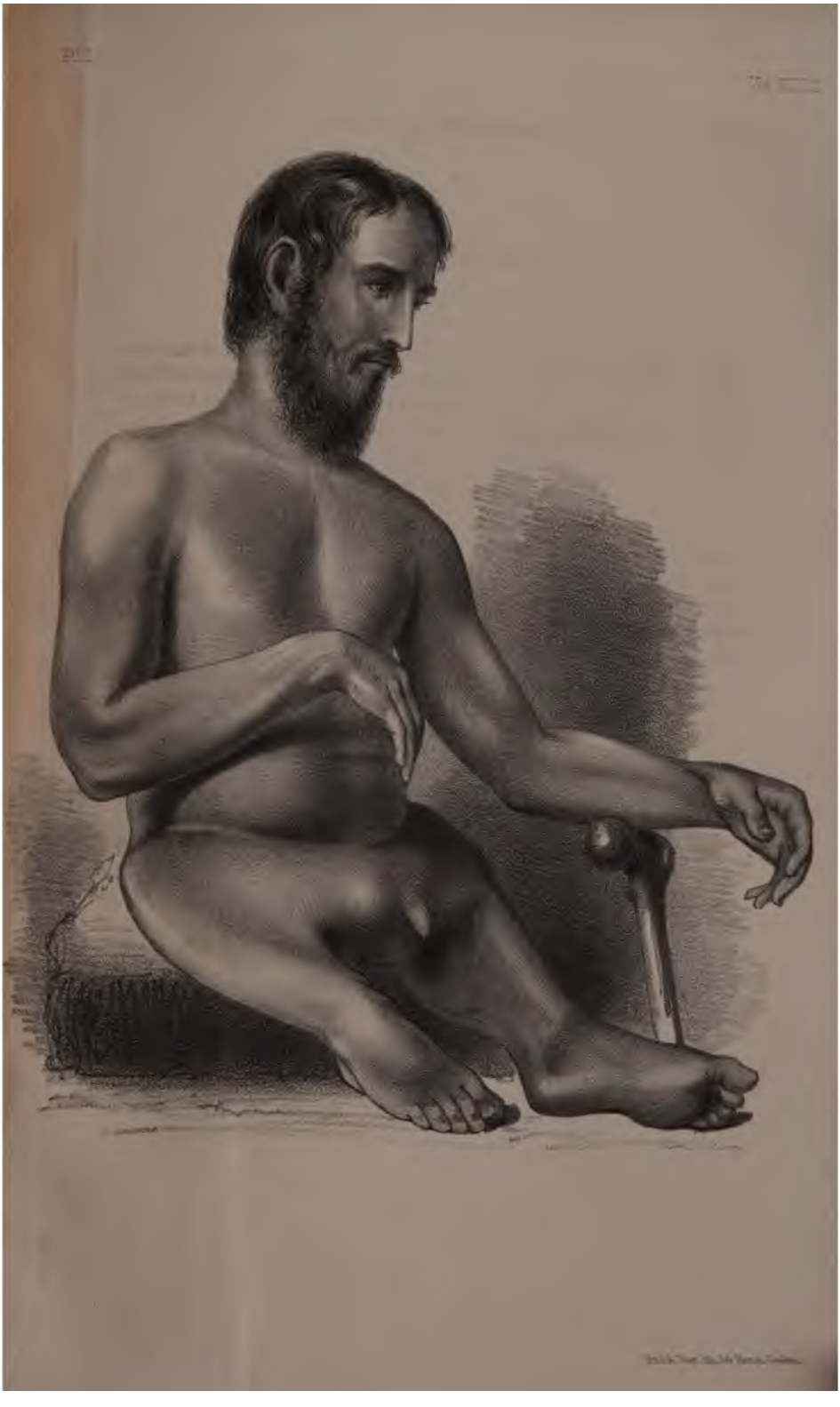
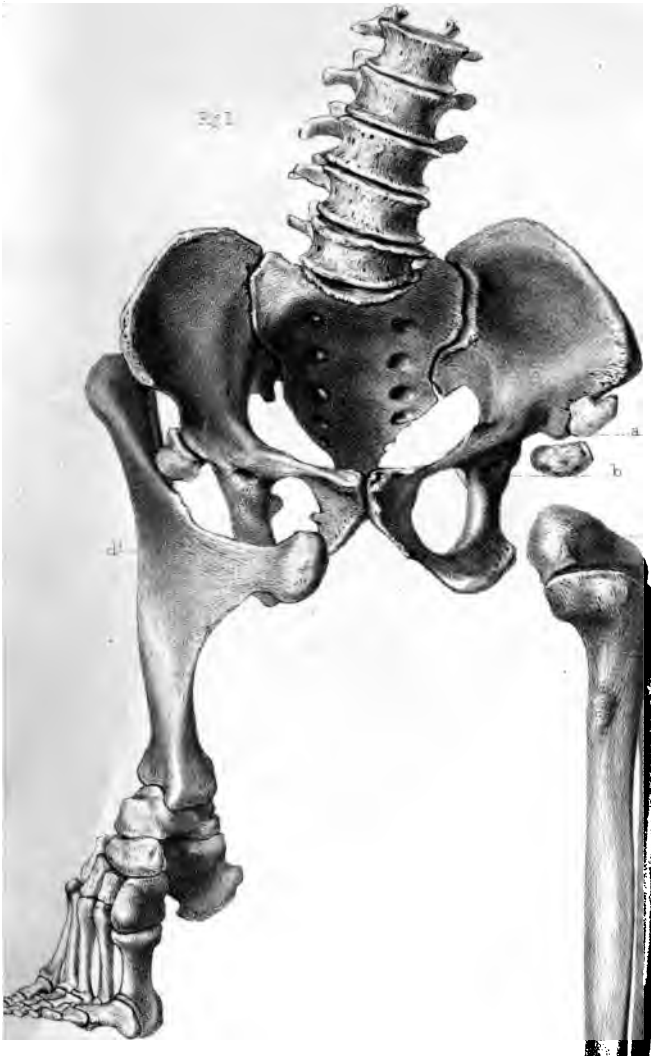
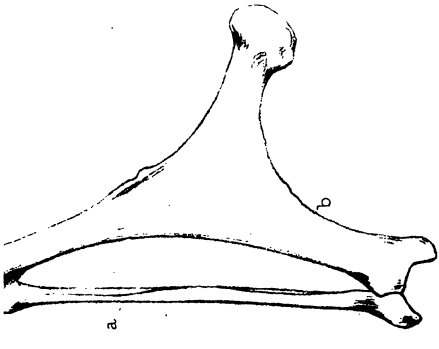


Fig 2

Chorion and coats of vessels studded with oil granules the nuclei of Chorion and of vessels much changed, and many obliterated 220 d.

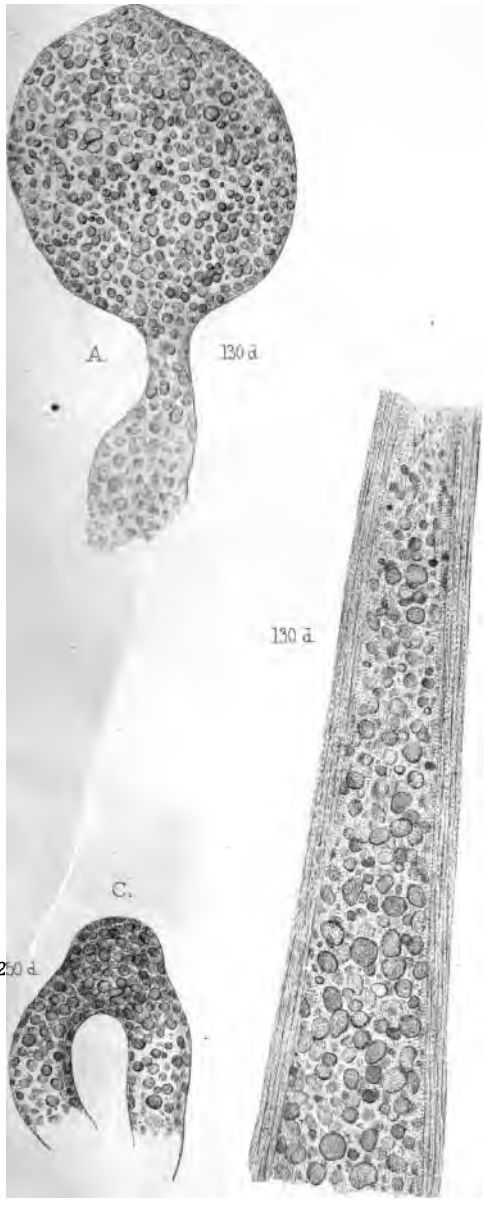


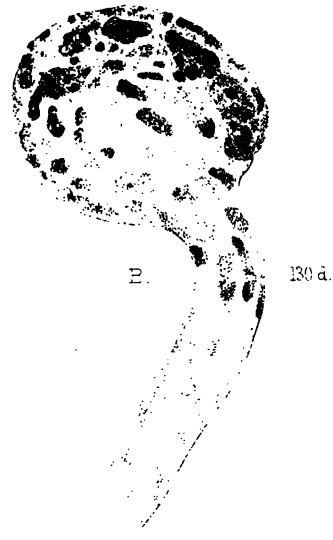


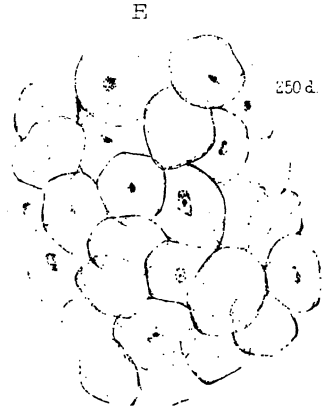


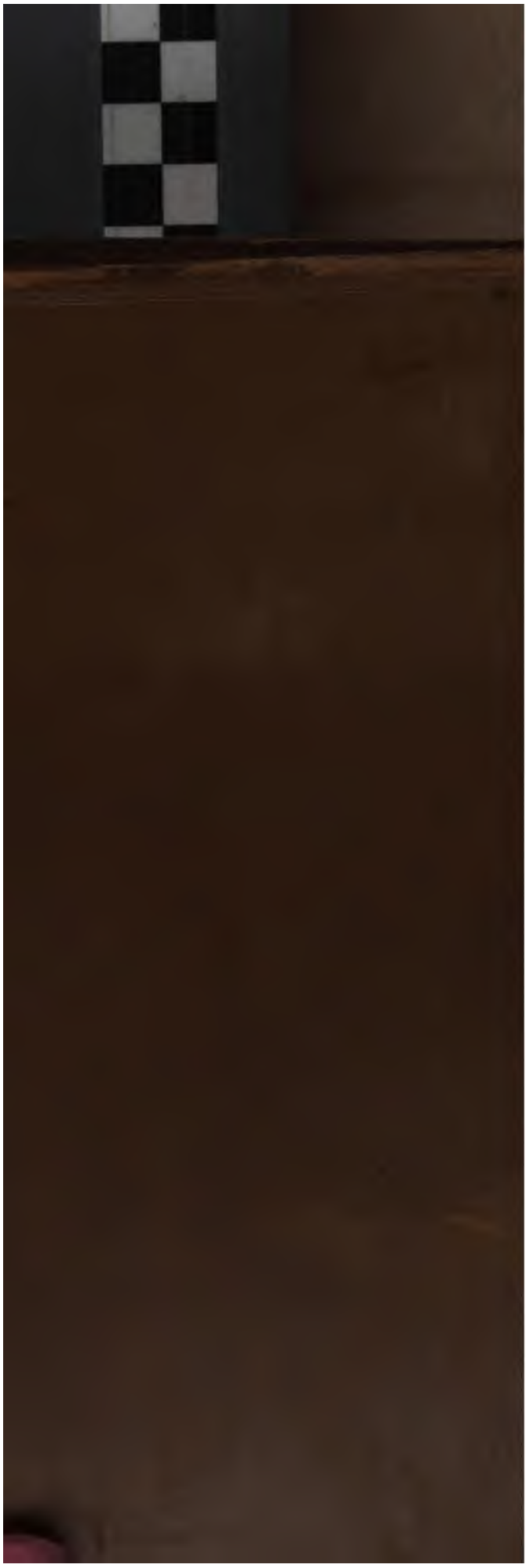
Ernst & Korn Verlag Berlin

Gill lithog. Edinburgh







A & B. Seminal tubes terminating in a dilated pouch. C. Lateral dilatation of a tube filled with dark granular matter. D. Uniform dilatation of a tube. E. Tessellated epithelium from a large cyst.

G H Ford & T West. sculp. Ford & West Lith. 54 Hatton Garden